

**An examination of the effects of stimulant medication on  
actual and self- perceived response inhibition: A  
comparison between children with and without attention  
deficit hyperactivity disorder**

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## **Declaration**

This thesis has been composed by myself and the work contained herein is my own

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## **Abstract**

**OBJECTIVE:** This study aimed to investigate whether methylphenidate medication was effective in improving response inhibition in children with ADHD, using a newly developed measure of response inhibition – the Animal Stroop Task. In addition, the study examined children's own attributions for their level of response inhibition across a variety of variables, including medication status.

**METHOD:** A between subjects design was used to compare a group of children with ADHD and a control group of normally developing children on measures of response inhibition, locus of control and behavioural attributions. Participants with ADHD were tested in two separate conditions – unmedicated and medicated. Additional within subjects comparisons were used to compare response inhibition and behavioural attributions across the two conditions.

**RESULTS:** There was no significant difference between the inhibitory control of children with ADHD and those of children in the control group but children with ADHD did show a significant improvement in inhibitory control following methylphenidate medication. There was no difference between the Locus of Control scores of children with and without ADHD and no significant relationship between locus of control and inhibitory control in any condition. Children with and without ADHD did not differ in the degree to which they attributed their performance to external factors and these attributions did not vary as a function of medication status.

**CONCLUSION:** The current findings contradict previous evidence documenting deficits in the inhibitory control of children with ADHD, and challenge the well-

established theory that poor response inhibition is the cardinal feature of ADHD. Reports of methylphenidate improving the functioning of children with ADHD are supported but there is no evidence to suggest that children with ADHD are any more likely to make external attributions for their behaviour, irrespective of medication status. Possible explanations for these findings are discussed and areas for future research suggested.

# 1. Introduction

## 1.1. General Overview

Attention Deficit Hyperactivity Disorder (ADHD) is a developmental disorder with the cardinal features of developmentally inappropriate levels of sustained attention, distractibility, hyperactivity, and impulse control (Barkley, 2000). It arises early in childhood, typically between 3 and 7 years of age and, being relatively stable, persists through adolescence and into adulthood in 30-70% of cases (Weiss & Murray, 2003). Increasing numbers of children are being diagnosed with ADHD and current estimates indicate that 3-5% of the American population has ADHD (Goldman et al., 1998) with similar proportions within the UK population of primary-school aged children (Taylor, 1999b). ADHD is known to have neuropsychological consequences that are evident from psychological tests and measures of school failure (Seidman et al., 1997). As such, the disorder is associated with both concurrent and long-term impairments in academic and social functioning (Johnston, 1998). Comorbidity is common in ADHD and a significant proportion of those diagnosed go on to develop delinquent activities and antisocial personalities (Barkley, 2000). Consequently, the burden of ADHD to affected individuals, to their families, and to society is considerable.

Moving away from the traditional, established view of a triad of impairment (inattention, hyperactivity and impulsivity), neuropsychological models now consider children with ADHD to have difficulties in terms of cognitive attention and

executive functioning deficits (e.g. Lawrence et al., 2002). More specifically, researchers have emphasized poor behavioural/response inhibition as the central impairment of the disorder (Barkley, 1990). Such contentions are supported by the findings of research demonstrating children with ADHD are impaired on neuropsychological tests of response inhibition (e.g. Shue & Douglas, 1992; Pennington & Ozonoff, 1996).

While the behavioural difficulties associated with ADHD are often managed with psychotherapeutic approaches, there is an absence of positive outcome studies (Brown and Levers, 1999). Instead, a large-scale multi-modal treatment study of children with ADHD demonstrated that the most effective treatment for ADHD was closely managed pharmacotherapy (MTA Cooperative Group, 1999). Despite several challenges to these findings (e.g. Owens & Hoza, 2003), well-titrated medical treatment for ADHD remains the treatment of choice (Hood et al., 2005).

Several authors (e.g. Bugental et al., 1977) have expressed concern that successful treatment with medication may have an adverse effect on children's self-perceptions and argue that it may lead them to attribute their behaviour to external factors (e.g. the drug) while viewing their own efforts/abilities as having a relatively minor role. It has been speculated that this may result in children becoming reliant on drugs to focus their attention and effort, meaning that when medication is discontinued, they are left feeling that they have no way of controlling their behaviour (Rosen et al., 1985). Existing evidence is varied. While some case studies (e.g. Rosen et al., 1985)



and indirect evidence suggests that medication does have these negative effects, others have found little evidence to support the notion that medication produces predominantly external, medication-related explanations for performance (e.g. Milich et al., 1989). While most studies examining the attributional responses of children with ADHD measure task performance using tests of sustained attention and/or assessing their social/behavioural conduct, little research has looked at the direct effects of medication on response inhibition.

The aim of this study is to examine whether methylphenidate is effective in improving response inhibition in children with ADHD, using a using a newly developed measure of response inhibition – the Animal Stroop Task (Wright et al., 2003). In addition, the study intends to examine children's own attributions for their level of response inhibition across a variety of variables, including medication status.

### **Terminology**

In this study, the term Attention Deficit Hyperactivity Disorder (ADHD) will be used to describe the syndrome of inattention, overactivity and inattention. While there are different terms available depending on the diagnostic system utilised, these systems ultimately describe very similar disorders. For the purpose of this study, then, the terminology used by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) (American Psychological Association, 1994) will be adopted.

‘Response inhibition’ and ‘inhibitory control’ are both terms used to denote the same facility and are therefore used interchangeably throughout this research.

## **Literature Search**

Electronic literature searches were undertaken using OVID and Cochrane based databases, in addition to a Google Search Engine. The following key words were used in combination as well as individually – Attention Deficit Hyperactivity Disorder / ADHD / Hyperactivity / Children / Executive Function / Response Inhibition / Inhibition / Impulsivity / Stroop / Methylphenidate / Stimulant Medication / Pharmacological / Behavioural Intervention / Psychosocial Intervention / Locus of Control / Attributions / Self-perceptions. Citations in relevant articles were also reviewed.

The introduction will begin with an examination of the definition of ADHD. This will be followed by an overview of the current procedure for diagnosing ADHD and the aetiology of the disorder. The next section will focus on the role of Executive Functioning in ADHD, moving on to look more specifically at response inhibition and neuropsychological theories of ADHD, before examining the current methods of treatment for ADHD. Finally, the introduction will end with a discussion of behavioural attributions and locus of control in children with ADHD.

## **1.2. Attention Deficit Hyperactivity Disorder**

Attention Deficit Hyperactivity Disorder (ADHD) is a developmental disorder characterised by developmentally inappropriate levels of four core features - sustained attention, distractibility, hyperactivity, and impulse control (Barkley, 1997). It emerges early in childhood, usually between the ages of 3 and 7 years old and, being relatively stable, persists through adolescence and into adulthood in 30-70% of cases (Weiss & Murray, 2003). The disorder is more commonly found in boys, outnumbering the incidence of ADHD in girls by between 4:1 (Gaub & Carlson, 1997) and 3:1 (Barkley, 1997). ADHD encompasses three subtypes - inattentive, hyperactive-impulsive, and combined ADHD (Rappley, 2005). Increasing numbers of children are being diagnosed with the condition and it is now one of the most commonly diagnosed behavioural disorders in children and adolescents (SIGN, 2001). While prevalence rates vary depending on the population sampled, as well as the method of assessment and diagnostic criteria used, most estimates sit between 5 and 10% (Taylor et al., 1991). Current estimates indicate that 3-5% of the American population has ADHD (Goldman et al., 1998) with similar proportions within the UK population of primary-school aged children (Taylor, 1999b). ADHD is known to have neuropsychological consequences that are evident from psychological tests (Seidman et al., 1997). As such, the disorder is associated with both concurrent and long-term impairments in academic and social functioning (Johnston, 1998) which can lead to frustration, emotional lability and low self-esteem amongst those diagnosed (Moser & Bober, 2002).

Comorbidity is common in ADHD. Sixty seven percent of children diagnosed with ADHD present with more than one psychiatric diagnosis, of which oppositional defiant disorder (33%), conduct disorders (25-35%), anxiety disorders (<25%) and learning disability (12%) are the most frequently cited (Moser & Bober, 2002). Co-morbid reading disorders are also particularly common in children with ADHD, with such difficulties occurring in approximately one-third of clinic referred children (August & Garfinkel, 1990). Although the rate of ADHD diminishes in adolescence (Cohen et al., 1993), the disorder does persist in a high proportion of individuals (Weiss & Murray, 2003). However, the resources available to such individuals are limited and a significant proportion go on to develop delinquent activities and antisocial personalities (Barkley, 2000). As well as the individuals themselves being affected, parents of children with ADHD experience higher levels of stress and depression and sibling relationships are also more troublesome (Hankin, 2001). Compared with the general population, patients with ADHD are more likely to require access to mental health services and to generate substantial costs in terms of resources, time and money across home, school and community settings (DeNisco et al., 2005). Consequently, the burden of ADHD to affected individuals, to their families, and to society is considerable (Barkley, 2000).

### **1.3. How is ADHD Diagnosed?**

The initial presentation of ADHD will usually be to general practitioners or other primary care, education or social work professionals. While all of these professions

may be involved in a preliminary assessment, it is recommended that the child is also referred for assessment by a child and adolescent psychiatrist or paediatrician specialising in this field (SIGN, 2001).

Although there are now a number of well-tested diagnostic interview methods available for the reliable assessment of ADHD, there is no independent, valid test for ADHD (NIH Consensus Statement, 1998). It has been suggested, however, that the critical components of any ADHD assessment should include a thorough history of the patient and their family (involving detailed patient and carer interviews), behavioural checklists and reports from both parents and teachers, a brief mental status examination, as well as a full physical examination and laboratory testing of various bodily functions – in order to exclude any underlying medical conditions and/or to identify co-morbid disorders (DeNisco et al., 2005).

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) (American Psychological Association, 1994), and the International Classification of Diseases, Tenth Edition (ICD-10) (World Health Organisation, 1992) currently provide two of the most widely used criterion to assist diagnosis. While many of the various systems use different terms to describe the disorder (e.g. Attention Deficit Hyperactivity Disorder; Hyperkinetic Disorder; Attention Problems Syndrome), most systems ultimately describe very similar syndromes, encompassing features of inattention, overactivity and impulsivity.

The diagnostic criterion outlined in the DSM-IV and ICD-10 is generally very similar, with the few differences that do exist relating to symptom severity and pervasiveness. Diagnosis of ADHD is usually not certain before the age of four years old, due to the fact that many of the identified behaviours may be typical of normal development (DeNisco et al., 2005). Nonetheless, some symptoms of hyperactivity, impulsivity or inattentiveness must be evident before the age of 7 years and have persisted for over 6 months (DSM-IV, 1994). These criteria differ slightly within the ICD-10 which stipulates an onset of before six years of age with symptoms being of 'long duration' (ICD-10, 1992). Appendix 7.1 contains the diagnostic criteria for attention and hyperactivity syndromes as outlined in the DSM-IV, ICD-10.

Both manuals specify that in order for a diagnosis to be reached, symptoms must be present in at least two different settings, such as home and school. Indeed, within the United Kingdom, the diagnosis of ADHD tends to be more narrowly defined, with much of the emphasis being placed on this required stability of problems across home and school environments (Carr, 1999). In contrast, this cross-situation consistency tends not to comprise a core diagnostic criterion in the US (Hinshaw, 1994).

Furthermore, in order to reach a diagnosis of ADHD, symptoms must have caused significant functional impairment and should not be better accounted for by other

mental disorders (e.g. pervasive developmental disorder, depression or anxiety, schizophrenia, other psychotic disorders).

The DSM-IV goes on to divide ADHD into three distinct subtypes – Inattentive; Hyperactive-Impulsive; Combined. The presence of six or more symptoms of inattention (e.g. difficulty sustaining attention, inability to listen when spoken to, easily distractible) can result in the diagnosis of ADHD, predominantly inattentive subtype (AD/HD<sub>in</sub>). On the other hand, a patient who displays six or more symptoms of hyperactive or impulsive behaviour (e.g. fidgeting, excessive talking, excessive running/climbing in inappropriate situations) would tend to be diagnosed with ADHD, predominantly hyperactive-impulsive type (AD/HD<sub>hyp</sub>). A diagnosis of ADHD combined type (ADHD<sub>com</sub>) is given when the symptoms are evenly divided (i.e. six or more symptoms in each category).

The ICD-10 favours the term ‘hyperkinetic disorder’, arguing that there is insufficient knowledge about the psychological processes to warrant identifying the disorder as ‘attention deficit’. ICD-10 tends to specify two different sub-types, depending on whether additional features of conduct disorder are present – ‘hyperkinetic disorder with disturbance of activity and attention’ when antisocial features of conduct disorder are absent; ‘hyperkinetic conduct disorder’ when criteria for both conduct disorder and hyperkinetic disorder are met.

In line with DSM-IV and ICD-10, there are numerous checklists and rating scales which aim to aid the diagnostic process. For instance, both the Conners ADHD index (Conners, 1996) and Achenbach's Child Behaviour Checklist System (Achenbach, 1991) utilise a parent and teacher report form containing similar sets of core features, with a number of additional items being specific to either the home or school contexts. Several similar rating scales are available, most of which discriminate adequately between children with and without ADHD (Guerva & Stein, 2001). In a recent review examining various diagnostic procedures (Green et al., 1999), the Conners ADHD index and DSM-IV-based symptoms scales (teacher and parent versions) performed best out of seven rating scales and four checklists. Barkley's school situation questionnaire (Barkley & Edelbrock, 1987) performed most poorly. The authors note, however, that having used single studies to calculate effect sizes for each checklist, reliability may be limited and results should therefore be interpreted with caution (Green et al., 1999).

#### **1.4. The Aetiology of ADHD**

While ADHD is recognised as a medical diagnosis, as we can see from the above diagnostic procedures, it does not refer to a set of biological symptoms with a distinct pathology. Instead, like the majority of psychiatric disorders, ADHD is a term used to describe a particular cluster of behaviours (Brown, 2004).

As such it is perhaps not surprising that, over the years, the classification of what constitutes ADHD has changed dramatically. Descriptions of ADHD date back to



the beginning of last century when Still (1902) identified a group of children he classed as having difficulties in 'moral control'. He believed the central deficit in this disorder was volitional inhibition. Subsequent descriptions moved onto identify hyperactivity, inattention and impulsivity as key to the disorder (Laufer & Denhoff, 1957), with a later emphasis being placed on self-regulation and inhibition as the cardinal features (Douglas, 1983; Barkley, 1997). Carr (1999) outlines the clinical features of ADHD as being within the domains of cognition, affect, behaviour, physical health and interpersonal adjustment. Within the domain of cognition, features include short attention span, distractibility, and inability to anticipate behavioural consequences which is usually accompanied by an inability to internalise rules of social conduct. With regard to affect, individuals usually display reduced levels of impulse control and tolerance for frustration. Low mood is common, but often accompanied with periods of increased excitability. In terms of behaviour, children with ADHD are typically very active and aggression, anti-social behaviour and increased levels of risk-taking behaviour are also common. Physical health problems may include injuries or medical complications which are often associated with antisocial behaviour such as fighting and/or drug abuse.

While the behaviours associated with ADHD are relatively easy to observe and measure, it has proven considerably more difficult to identify particular cognitive delays, deficits or dysfunctions that might underpin the disorder (Warner-Rogers, 2002). Indeed, the origins of ADHD have been, and continue to be, much debated (see Barkley, 1990 or Tannock, 1998 for a review), with several possible aetiologies

being put forward - including both environmental and central nervous system disturbances (e.g. Rappley, 2005), as well as more genetic based explanations (e.g. Moser & Bober, 2002).

It is now generally recognised that the development of ADHD is likely to be multifactorial (Taylor, 1999a), and, involving a combination of interacting genetic, biological and environmental factors, it has been referred to as

*“a paradigm for a true biopsychosocial disorder”* (Tannock, 1998, pg. 65).

#### **1.4.1. Genetics of ADHD**

There is currently little doubt that genetic factors play a substantial role in the development of ADHD (Stevenson, 2005). Initially, studies focused on demonstrating the highly heritable nature of ADHD. For example, children with ADHD are more likely to have a parent with the disorder (Smalley et al., 2000), and figures show a higher concordance rate of pervasive hyperactivity among monozygotic twins (51%) in comparison to dizygotic twins (30%) (Goodman & Stevenson, 1989 a, b).

From here, studies moved their focus to identifying which particular genes might be involved. While several potential genetic mechanisms continue to be explored, research has primarily focused on the genes involved in dopaminergic transmission, with particular attention having been given to exploring variations in the dopamine 4

receptor gene (LaHoste et al., 1996). More specifically, current consensus suggests that inherited variants of the genes that function to modulate dopaminergic neurotransmission may play a role in changing the structure and function of particular brain regions, subsequently leading to abnormalities in psychological functioning, such as difficulties inhibiting inappropriate responses (Taylor, 1999a). These contentions are backed up by findings that dopaminergic drugs (methylphenidate) are clinically effective in reducing the core symptoms of ADHD, while imaging studies utilising Positron Emission Tomography (PET) and Magnetic Resonance Imaging (MRI) techniques have highlighted an association between ADHD and the frontostriatal circuitry - an area rich in dopaminergic activity (Daley, 2006). Nonetheless, genetic studies continue to explore new avenues, such as the role played by serotonin transporters (Manor et al., 2001), and now extend to examining the possibility that different genetic mechanisms may be involved dependent on the specific subtype of ADHD being investigated (Daley, 2006).

However, genetic studies clearly cannot provide a full explanation of the development of ADHD. As yet, no single or multiple gene association has been identified (Rubia & Smith, 2001). Furthermore, although there is substantial evidence for a familial transmission in ADHD, this link cannot exclude an explanation of a purely environmental causation. Family and twin studies are compounded by the increased incidence of shared environmental experience, while adoption studies are limited and subject to methodological limitations (e.g. the selective placement of adoptees, the lack of direct comparison between the biological

and adoptive relatives of the same adoptee, as well as failure to perform blind evaluations of their participants (Joseph, 2000)). As such, the potential role of genetic factors in ADHD is an issue that remains to be confirmed.

#### **1.4.2. Brain Structure in ADHD**

There is now an abundance of recent research examining the anatomical structure of the brains of children with ADHD. Functional and structural neuroimaging studies suggest that there are abnormalities within the brain circuits linking the prefrontal cortex, striatum and cerebellum (Castellanos & Acostas, 2002) and, in comparison to controls, children with ADHD tend to have slightly lower total brain volumes (Castellanos et al., 1996). However, the results remain conflicting with regard to the exact pathology of the disorder (Eliez & Reiss, 2000) and several authors (e.g. Daley, 2006, Rubia & Smith, 2001) have highlighted the need for further research in this area. Notably, Rubia & Smith (2001) also caution that, given recent findings that function, behaviour and environmental stress factors have been shown to alter brain structure and function, neurobiological abnormalities can no longer be seen as the cause of the disorder.

#### **1.4.3. Environmental Influences on ADHD**

Warner-Rogers (2002) highlights that genetic research has also revealed a number of non-genetic factors which can impact on the developmental course of ADHD. For instance, maternal smoking and pre-eclamptic toxemia have been associated with hyperactivity (Taylor, 1999a), as has prenatal exposure to alcohol (Taylor et al,

1991). Other risk factors for the development of ADHD include very low birth weight, severe anoxia, and early lead poisoning (Taylor, 1999a).

While problems in family functioning may not give rise to ADHD symptoms per se, it can affect the development of such problems (Taylor, 1999a). Indeed, the interaction between genetic and environmental factors is highlighted by contentions that children with a genetic predisposition will present with the disorder when placed in the right environment, typically one of chaotic parenting (Johnston & Mash, 2001). However, it should be noted that whilst studies have demonstrated less optimal parenting styles in parents of children with ADHD (e.g. Gardner, 1994), the relationship between ADHD and parenting may result from a combined force of negative aspects of the child influencing the parents behaviour, as well as negative features of the parents affecting the child's behaviour (Daley, 2006). In addition, when we consider the aforementioned familial link between children with ADHD having a parent with ADHD (Smalley et al., 2000), we can appreciate how, in some families, parents' own symptoms may interfere with any attempt to implement a suitably consistent approach to parenting.

Diet is another factor which often comes under any discussion around the environmental variables influencing ADHD. However, while symptoms of ADHD have been associated with food additives, refined sugars and fatty acid deficiencies, the validity of such studies is troubled by numerous methodological problems and small sample sizes (Schnoll et al., 2003). More recent studies have failed to make

links between hyperactivity and food additives (e.g. Bateman et al., 2004) and current guidelines on ADHD in Children and Young People stipulate that there is no established evidence base allowing specific dietary therapies to be recommended (SIGN, 2001).

Overall then, it appears that the developmental course of ADHD may be subject to a number of interacting genetic, biological and environmental factors. However, despite various associations having been made, results are often conflicting and, as yet, a fully-comprehensive aetiological explanation for the disorder remains to be established. As such, this lack of clarity, serves to add to the controversy surrounding the disorder as a whole.

Barkley (1997) highlights that, historically, research has been based on exploratory and descriptive methods, and, as such, the widely accepted view of ADHD (as stipulated by DSM-IV) has primarily focused on behavioural deficits – i.e. inattention, hyperactivity and impulsivity. He argues that these features are unable to adequately account for the full range of cognitive and behavioural deficits typical of ADHD and draws attention to a number of more theory-driven neuropsychological explanations which aim to give a more comprehensive explanation. Thus, moving away from the traditional, established view of a triad of impairment (inattention, hyperactivity and impulsivity), the more recent neuropsychological views tend to regard children with ADHD as having difficulties within the realms of self-regulation and executive functioning (e.g. Lawrence et al., 2002; Barkley, 1997).

### 1.5. Executive Functioning in ADHD

‘Executive functioning’ is a term used to describe the mental activities that enable self-control and goal-directed behaviour. It is defined as:

*“those capacities that enable a person to engage successfully in independent, purposive, self-serving behaviour”* (Lezak, 1995, pg. 42).

Such abilities are thought to be regulated by the prefrontal cortex region of the brain, including its extended networks (Lezak, 1995). The term ‘executive functioning’ is regarded as encompassing several different processes, such as working memory, self-regulation of emotion, motivation and response inhibition (Barkley, 1997) and has been branded as,

*“the seat of social intelligence”* (Barkley, 2000, pg. 1068).

Executive impairment typically results in reduced capacity for self control and/or self-direction, heightened irritability and excitability, impulsivity, erratic carelessness, rigidity and difficulty in making shifts in attention and on-going behaviour (Lezak, 1995). Thus, in many ways the clinical description of executive impairment resembles the aforementioned behavioural descriptions of ADHD.

Indeed, due to the similarity in the presentation of individuals with ADHD and patients who have suffered frontal lesions in the brain - the latter also resulting in hyperactive, distractible and/or impulsive behaviour (Fuster 1989; Stuss & Benson,

1986) - a number of researchers have implicated frontal lobe dysfunction and executive impairment in ADHD (e.g. Rosenthal & Allen, 1978). Accordingly, neurological studies involving patients with ADHD have found evidence of neuroanatomical differences suggestive of anterior dysfunction (Hynd et al., 1993). Furthermore, instead of displaying the normal right-dominant frontal asymmetry, the brains of children with ADHD are typified by a smaller right frontal width, resulting in symmetrical frontal lobes (Hynd et al., 1990).

In line with these contentions, studies utilising tests of executive functioning have found that, when compared with matched controls, children with ADHD show deficits in a number of executive functioning tasks. For instance, Gorenstein et al. (1989) found deficits in attentional set shifting (Gorenstein et al., 1989), as well as deficits in response inhibition (Gorenstein et al., 1989; Booth et al., 2005), time slowness (Gorenstein et al., 1989) and planning ability (Weyandt & Willis, 1994).

In a recent review by Pennington and Ozonoff (1996), 15 out of 18 studies found a significant difference between ADHD participants and controls on at least one measure of executive function. Results yielded a number of tests which were particularly sensitive to ADHD, namely the Tower of Hanoi (assessing planning ability), Stroop (task of inhibition), Matching Figures Test (a measure of impulsivity) and the Trail Making Test, Part B (assessing flexible set-shifting). Consistent group differences were also found on measures of motor control and working memory (Pennington & Ozonoff, 1996).



Others, however, have failed to replicate these findings. For instance, using comparable measures of executive functioning, Grodinsky & Diamond (1992) found that participants with ADHD showed no significant impairments in attentional set-shifting, response inhibition or time slowness. More recent studies have also failed to replicate findings of deficits on motor planning and verbal fluency (Speltz et al., 1999).

It has been suggested that the equivocal nature of findings is perhaps due to the possibility that, having been validated on adults, many tests of executive function may have limited validity for use with children (Kempton et al., 1999). Nonetheless, considering the lack of clarity of results, it is perhaps not surprising that there is still debate surrounding the exact nature of these deficits (Pennington & Ozonoff, 1996) and much of the current research now focuses on identifying those executive functioning processes which are principal in explaining ADHD.

#### **1.6. Response Inhibition in ADHD/Neuropsychological Theories of ADHD**

Extending this work on executive functioning, Barkley (1997) argues that the central impairment in ADHD is a pervasive deficit in response inhibition – i.e. the ability to

inhibit a prepotent response<sup>1</sup>, interrupt an ongoing sequence and resist interference (Barkley, 1997). Response inhibition is defined more simply as,

***“the ability to stop (suddenly and completely) a planned or ongoing thought and action”*** (Williams et al., 1999, pg. 205).

This level of control is crucial in numerous everyday situations where changes to one’s surrounding environment result in pre-planned or ongoing actions becoming unexpectedly inappropriate (e.g. a person must stop crossing the road if a previously clear road suddenly becomes occupied by a speeding car). While results have been mixed (e.g. Oosterlaan & Sergeant, 1998, Schachar & Logan, 1990), the ability to inhibit responses has been found to improve throughout childhood (Williams et al., 1999; Simpson & Riggs, 2005)<sup>2</sup>, with the most rapid improvements in inhibitory control being between the ages of 3½ - 5 years (Simpson & Riggs, 2005). Improvements in inhibitory control have also been associated with increasing affect and behavioural regulation (Denckla, 1995).

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<sup>1</sup> Barkley (1997) defines a prepotent response as “that response for which immediate reinforcement (positive or negative) is available or has been previously associated with that response” (Barkley, 1997, pg 229).

<sup>2</sup> Furthermore, Barkley (1997) contends that response inhibition is one of the earliest emerging control processes. Williams et al. (1997) offer further support for such contentions, highlighting that given the significance of inhibitory control for survival, this developmental pattern would make particular sense from an evolutionary perspective.

### **1.6.1. Model of Response Inhibition**

Barkley (1997) contends that in ADHD it is the overall impairment in response inhibition that leads to secondary deficiencies in four other executive functions (i.e. working memory, internalisation of speech, self-regulation of affect-motivation-arousal, and reconstitution), as all are dependent on response inhibition for their execution. He presents this theory within a hierarchical model, where deficits in response inhibition - positioned at the top of the model - give rise to difficulties in these other, lower-order executive functions. This model is outline in Figure I.

Self-regulation refers to any self-directed action that functions to alter the probability of a subsequent response in order to modify the likelihood of a future consequence (Barkley, 2000). Given each executive function can be viewed as a type of self-directed action, self-regulation can be seen as an inherent part of executive functioning. Thus, effective inhibition is required not only for the control of these four secondary executive functions but for self-regulation as a whole.

Many of these self-regulation behaviours are observable in early development but, as the child's cognitive skills progress, these actions may become more private or internal in nature. Self-regulation may include skills such as the organisation of behaviour across time, self-directed speech and the ability to take rules or plans into account (Barkley, 1997). Thus, resisting temptation, delaying gratification and executing novel responses are all actions likely to require skills of self-regulation, and, as such, it is viewed as an inherently future-orientated process (Barkley, 2000).

Children with ADHD are considered to have problems with self-regulation - this often presenting as a tendency for them to be influenced by their immediate environment and imminent consequences (Barkley, 1997). In contrast, children without ADHD are more influenced by internal information such as past experience, rules and plans.

Given effective inhibition is regarded as essential in instigating its related executive functions and self-regulation, it follows that Barkley considers inhibition as the central deficit of ADHD. He argues that poor sustained attention (once itself seen as the crux of the disorder) should be seen as a secondary symptom. He claims a deficit in sustained attention is likely to be due to an underlying impairment in goal-directed persistence arising from poor inhibition and its associated effect on self-regulation (Barkley, 1997).

This model tends to dominate a lot of contemporary theory of ADHD and, as such, the view of response inhibition as the central deficit of ADHD is one which is now wide-spread (Barkley, 1997; Kerns et al., 2001).

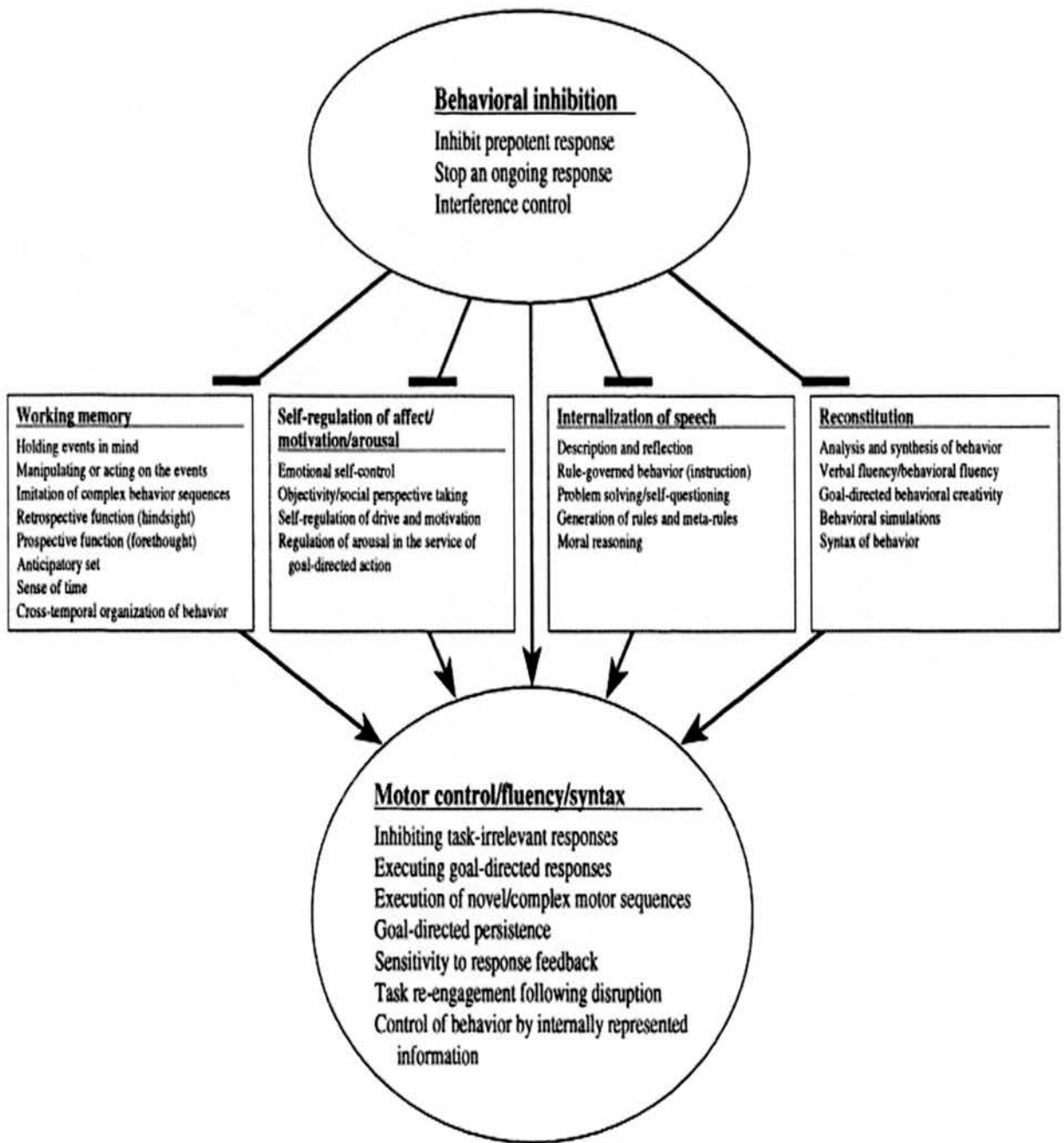


Figure I. A schematic configuration of a conceptual model linking behavioural inhibition with the performance of the four executive functions that bring motor control, fluency, and syntax under the control of internally represented information (Barkley, 1997).

### 1.6.2. An Alternative Model of ADHD

It may be worth noting that, relatively recently, research has emerged to challenge the dominant notion of response inhibition as the core deficit in ADHD. Sonuga-Barke et al. (1994) argued that most of the evidence supporting ADHD as a result of cognitive dysregulation was confounded by delay. Sonuga-Barke and her colleagues (1992) found that when children were asked to choose between a small immediate reward and a large, delayed reward, children considered as hyperactive, were more likely to choose the smaller immediate reward. However, such strategies were only chosen if they reduced the overall delay period. When choosing the small reward resulted in the same overall delay period as choosing the larger, postponed reward, hyperactive children were able to wait as well as the group of control children.

Furthermore, while children with ADHD were consistently found to make more impulsive responses and more errors on the matching figures test, Sonuga-Barke et al. (1994) pointed out that these studies were effected by trial constraints (i.e. as soon as one trial ended another began) and, as such, were confounded with delay. When these experiments were repeated in such a manner that early or impulsive responses had no influence on delay, the responses from children with ADHD were comparable with that of controls. Such findings lend support for a 'delay aversion' hypothesis (Sonuga-Barke et al., 1996) in which it is argued that the cognitive deficits thought to be shown by children with ADHD could actually be more motivational in nature – i.e. children with ADHD are averse to delay. Sonuga-Barke et al. (1996) contend

that the influence of delay on behaviour is dependent on whether or not the child is in control of their environment. When the child is in control of their environment, they can opt to minimise delay by acting impulsively e.g. jumping a queue at the slide (Daley, 2006). Conversely, when a child is not in control of their environment, or expected to conform to certain behavioural rules, they may seek to reduce the *subjective* experience of delay (e.g. by day-dreaming, fidgeting etc.). Although recent research by Kerns et al (2001) also found group differences on a measure of delay-aversion, this effect did not remain significant after controlling for conduct problems, leading the authors to suggest that most, if not all, of the association between hyperactivity and delay aversion can be explained by co-occurring conduct problems, rather than ADHD per se (Kerns et al., 2001).

Thus, despite the emergence of these relatively new concepts, the literature generally remains dominated by theory citing response inhibition as the central deficit of ADHD (Barkley, 1997).

### **1.6.3. Neuropsychological Tests of Response Inhibition**

Accordingly, consistent and robust evidence exists to suggest that children with ADHD do show deficits in tests of response inhibition. Several studies have shown that, in comparison to controls, children with ADHD show marked impairments in inhibitory responses across go/no-go tasks (Booth et al., 2005; Hartung et al., 2002; Charman et al., 2001; Iaboni et al., 1995; Shue & Douglas, 1992), the continuous performance task (Halperin et al., 1990), the stop signal task (Schachar & Logan,

1990) and Stroop interference tasks (Gorenstein et al., 1989; Grodinsky & Diamond, 1992; Houghton et al., 1999).

For instance, Shue & Douglas (1992) assessed response inhibition in children with ADHD aged between 8-12 years, using the Go/No-go task. This assessment acts as a simple measure of motor inhibition, whereby participants are required to press a response key as quickly as possible when presented with a “go” signal (S+), but need to refrain from pressing in response to a “no-go” signal (S-). Responses to the No-go signal are scored as errors. Results revealed that children with ADHD made significantly more errors than control children.

In a review of eighteen studies of executive functioning in ADHD, Pennington & Ozonoff (1996) concluded that executive functioning deficits are consistent in ADHD. They went on to highlight the measures that were found to be most sensitive to ADHD – i.e. the Tower of Hanoi (assessing planning ability), Stroop (task of inhibition), Matching Figures Test (a measure of impulsivity), the Trail Making Test, Part B (assessing flexible set-shifting), in addition to ‘purer’ measures of motor inhibition (e.g. Go/No-go, Stop tasks, NEPSY Inhibition etc). Thus, with a high proportion of these ‘most sensitive’ tests tapping processes of inhibition, measures of inhibition would appear to be particularly sensitive to ADHD.

These contentions are backed by functional magnetic resonance imaging (fMRI) data. For instance, Booth et al. (2005) assessed both visual selective attention (using



a task requiring a visual search of a conjunction target in a field of distracters) and response inhibition (measured with a go/no-go task). While there were limited group differences in the selective attention task, there were large group differences in the response inhibition task. In comparison to the brains of control children, children with ADHD showed significantly decreased activation in the fronto-striatal regions of interest – the area of the brain considered as the neural basis of response inhibition (Booth et al., 2005). The authors interpret these findings as evidence to suggest that children with ADHD are unable to effectively engage the fronto-striatal brain network to maintain appropriate behaviours or inhibit inappropriate behaviours, and concluded that results were consistent with the hypothesis that response inhibition is a specific deficit in attention deficit hyperactivity disorder (Booth et al, 2005).

Similar findings have also been obtained by Rubia et al. (2001). Using a more comprehensively modified test of inhibitory control (i.e. the Maudsley Attention and Response Suppression (MARS) battery, (Rubia et al., 2001)) children with ADHD were impaired in the ‘more demanding’ inhibition tasks such as the go/no-go, stop and reversal tasks – all of which required the inhibition of discrete motor responses. This was further corroborated by decreased activation in the right prefrontal brain regions of those with ADHD.

Thus, there appears to be a wealth of evidence offering support for a response inhibition deficit in children with ADHD, the majority of which appear to be measured using ‘Stop’ or ‘Go/NoGo’ paradigms. Indeed, Oosterlaan et al. (1998) conducted a recent meta-analysis of eight studies which used the Stop Task to

examine response inhibition - incorporating a total of 456 children aged 6-12 years. In seven of the eight studies considered, children with ADHD demonstrated flatter inhibition functions, indicating significantly poorer response inhibition than controls. As such, the authors concluded that consistent and robust evidence had been found to support contentions that poor response inhibition was at the core of the disorder and claim that this finding may extend to account for the central deficit of other externalising disorders (e.g. Conduct Disorder) (Oosterlaan et al., 1998). Findings have recently been extended from studies looking exclusively at children, and there is now additional support for a core deficit in behavioural inhibition in *adults* with ADHD (Bekker et al., 2005).

It should be noted, however, that while the majority of studies appear to measure inhibition using 'Stop' or 'Go/NoGo' paradigms there may be some methodological limitations of these tasks. For instance, such tests depend on the establishment of a 'well-learned' response (e.g. a simple motor response to a visual/auditory signal), prior to inducing the inhibition of that response. However, simple reaction times may not be 'well-learned' and, as such, might not necessitate demands on inhibition in order to prevent the response (Wright et al., 2003). In addition, inhibitory function is believed to have a more general cognitive basis. Thus, many pathological and developmental changes in inhibitory function are often associated with a broad range of associated behavioural symptoms such as language and social dysfunction (Wright et al., 2003). As such, tests that use motor response as a single measure of inhibition may have limited value.

Nonetheless, results demonstrating impaired response inhibition in ADHD are not always consistent. For instance, a recent study by Kerns et al. (2001) tested response inhibition in children diagnosed with ADHD using both a visual version of the Go/No Go task alongside the 'Golden' version of the Stroop task (Golden, 1978) - a measure of verbal inhibitory capacity. Analysis of results showed that only some measures of inhibition revealed group differences - i.e. children with ADHD did make a significantly higher number of omission errors on the Go/NoGo Task. However, with regard to both the rate of commission errors in the Go/NoGo task and the interference measure of the Stroop task, the performance of children with ADHD was comparable to that of controls. The authors present a number of possible explanations for these results. Most notably, they highlight the aforementioned contentions of Barkley (1997), which suggest that rather than inhibition representing a unified construct, it may be more appropriately conceived as several components working in unison (Barkley, 1997). Based on these premises, Kerns et al. (2001) propose that not all levels of inhibition may be impaired in children with ADHD. Hence, their performance on tests may vary according to which component of inhibition is being tapped.

Such contentions are interesting when considering the findings of a recent meta-analysis, aimed at examining studies of inhibition which utilise the Stroop task (van Mourik et al. 2005). A total of seventeen studies, encompassing 1395 participants aged between 6 - 27 years, were analysed in an attempt to determine the extent of any deficits in interference control in children and young adults with ADHD. In the

Stroop Colour-Word Test (Stroop, 1935), participants are presented with the words of colours (e.g. red). However, these words are printed in an opposing colour of ink (e.g. blue). Participants are asked to name the colour of ink in which the word is printed. Thus, the automatic reading response must be inhibited, while the colour of the ink is named (Scheres et al., 2003). The authors concluded that, with deficits either being completely absent or very small, the standard form of the Stroop Colour-Word task does not provide strong evidence for a deficit in interference control in children with ADHD.

However, whilst alleging to use the traditional method of calculating the interference score in order to yield a 'purer measure' of interference, the authors do acknowledge that the findings were strongly influenced by the particular type of scoring method. They also highlight the possibility that the standard Stroop Colour-Word Task may not serve as a valid measure of interference control in ADHD. For instance, while the current analysis looked only at studies utilising the standard card-based version of the task, they contend that a 'trial-by-trial' computerised version of the task may allow greater sensitivity to attentional pathology, as well as enabling more accurate measurement of response times and variability. Indeed, when using a computerised version of the Stroop, differences in reaction times have been detected between children with ADHD and controls (Carter et al., 1995). However, these results failed to be replicated in a subsequent study (Gaultney et al., 1999).

In addition, numerous other studies have made use of the Stroop Task to demonstrate evidence of a deficit in response inhibition in children with ADHD (e.g. Houghton et al., 1999; Grodinsky & Diamond, 1992). Indeed, although unclear which versions of the Stroop were employed, Pennington & Ozonoff (1996) referred to the Stroop as one of the measures which demonstrated the most consistent impairments in children with ADHD. Furthermore, subsequent discriminant analysis revealed that the Stroop was one of the most consistent measures in discriminating children with ADHD – yielding significant discriminating power in all three of the studies assessed (Pennington & Ozonoff, 1996).

VanMourik et al. (2005) highlight reasons why Stroop-type tasks may yield conflicting results. For instance, children with ADHD have been observed to have naming deficiencies (Tannock et al., 2000). As such, their lower score may be due to slower rapid naming instead of poor interference control. Additionally, not all studies control for reading ability. This is especially relevant given the high rates of co-morbidity between ADHD and reading disorder (e.g. August & Garfinkel, 1990). If a child cannot read well, it will likely be easier for them to ignore word-meaning which could potentially lead to relatively faster responses and less interference in kids with co-morbid ADHD and reading difficulties. Although vanMourik et al. (2005) found no significant difference in the interference score between children with ADHD and children with ADHD and a comorbid reading disorder, the previously described methodological weaknesses may have been masking such effects.

Such concerns have been addressed by more recent, alternative versions of the Stroop-task. These adaptations tend to have the same underlying principle as the original Stroop-task but present their stimuli in the form of pictures. A number of such measures are now available for use e.g. the Day-Night Task (Gerstadt et al., 1994) as well as the 'Fruit Stroop' task (Archibald & Kerns, 1999). In addition, Wright et al. (2003) recently presented a new, pictorial 'Animal Stroop' measure of inhibitory function which appears to be appropriate for use with younger children, and those with reading difficulties (Wright et al., 2003). What's more, within a school-based sample, initial research suggests that the Animal Stroop appears to identify those at risk of hyperactive symptomatology (Wright et al., 2003).

Overall then, while there is a strong neuropsychological evidence-base for a response inhibition deficit in ADHD, findings are mixed and may depend on the type of measure used. It may be possible that certain methodological limitations, both of the studies' design and of the tests used, are responsible for the inconsistent nature of results. The possibility that different tasks tap distinct components of inhibition - which may or may not be affected by ADHD (Kerns et al., 2001) – might also offer a potential explanation for the discrepancy of findings.

### **1.7. Current Treatment of ADHD**

A wide range of pharmacological and non-pharmacological therapies have been tried and continue to be used to treat ADHD, including one-to-one therapy, dietary

interventions, allergy treatments, biofeedback, play therapy etc. (Pelham et al., 1998).

However, many have failed to demonstrate their effectiveness as a treatment for ADHD. For instance, current guidelines on ADHD in Children and Young People stipulate that there is no established evidence base allowing specific dietary or alternative therapies to be recommended (SIGN, 2001).

Within the literature, there is a general consensus that only three treatments have been validated as effective *short-term* treatments for ADHD – behaviour modification, central nervous system stimulants and a combination of these therapies (Pelham et al., 1998).

#### **1.7.1. Psychotherapeutic Interventions for ADHD**

The extensive use of psychotherapeutic approaches in managing the behavioural difficulties associated with ADHD has generated a lot of investigation examining the effectiveness of such programmes. Some of these techniques focus on the child's parent and/or teacher while others concentrate on the child themselves. Such techniques might include educating parents and teachers about the nature of ADHD in order to facilitate understanding and promote realistic expectations of the child, providing them with simple strategies to reduce behaviour problems by adapting the child's environment and training them in behaviour-management skills. Direct interventions with the child may involve teaching the child skills of self-control and

self-monitoring and may have quite specific aims e.g. attention training or anger management.

While the effectiveness of programmes aiming to train parents in behavioural management techniques varies across individuals, they have been shown to reduce conflicts and non-compliance in children with ADHD (Barkley et al., 1992). Indeed, applying Task Force Criteria for empirically supported treatments, Pelham et al. (1998) concluded that both behavioural parent training and classroom-based behavioural interventions were effective in treating ADHD. The authors went on to separate the parent and classroom interventions into four categories – clinical behaviour therapy; direct classroom management, cognitive behavioural intervention and intensive packaged behavioural treatments. Overall, results suggested that cognitive behavioural treatments do not provide clinically significant improvements in the behaviour or academic performance of children with ADHD. It is worth noting that this conclusion contrasts with previous meta-analyses where cognitive interventions have been deemed as effective in treating impulsivity (Baer & Nietzel, 1991). However, such research was laboratory based and did not involve diagnosed and/or clinically referred children, thus restricting the generalisability of results. On the other hand, behaviour modification or contingency management showed considerable benefits over all studies assessed (Pelham et al., 1998).

A number of limitations of this study should be noted. The authors failed to outline the details of the requirements on which they were assessing the studies and also



gave few details regarding their search strategy or inclusion criteria. Such shortcomings have led other researchers to question whether important studies may not have been included (Guevara & Stein, 2001). Furthermore, the studies reviewed only covered children of elementary or pre-school-age, thus findings may not be generalisable to adolescents or adults with ADHD (Pelham et al., 1998)

Certainly, the research examining the outcome of psychotherapeutic research is mixed and often overshadowed by the wealth of research looking at pharmacological techniques. For example, a recent review by Klassen et al. (1999) concluded that behavioural therapies alone were not efficacious. Although not more efficacious than pharmacological treatment, combination therapy (i.e. behavioural therapy and pharmacological therapy) was found to be more effective than no treatment, placebo and behavioural therapy alone. However, improvements in behaviour were perceived by parents only and did not extend to the views of the teachers.

Despite reaching these conclusions, the authors do draw attention to the fact that their findings are somewhat limited by the imbalance of treatment modalities included in their review. Out of a total 26 papers examined, only two of the studies looked at behavioural treatments, with a further three assessing combined treatments. All of these studies were considered relatively poor methodologically and all were based on only small sample sizes. Nonetheless, reviews such as these, often lead to researchers concluding that there is an absence of positive outcome studies with regard to the effectiveness of psychotherapeutic approaches in the treatment of

ADHD (Brown and Levers, 1999). Furthermore, even when these programmes do appear to have some success, improvements tend to be temporary and not generaliseable to more natural environments (Hinshaw et al., 1998).

The validity of research examining psychotherapeutic approaches is often compounded by a number of factors. Unlike evaluations of stimulant medications, it is much more difficult to run highly controlled experimental studies looking at behavioural treatments. Children with ADHD demonstrate various combinations of problems and often differ in the way they present to clinicians, parents, teachers and their peers. As such, needs vary depending on the individual child. However, the research literature combines these ADHD cases by convenience and assesses predetermined therapy programmes without considering individual needs and differences. It may be that such mechanisms can account for the weak and inconsistent findings for psychotherapeutic types of interventions (Klassen et al., 1999). Furthermore, many of these studies are often limited to short-term evaluation and fail to follow-up the child or extend investigations into their natural environments (SIGN, 2001).

It should be noted that while there may be a lack of evidence to support psychotherapeutic techniques with regard to treating ADHD symptoms per se, combination therapies are often used in the treatment of co-morbid conditions and psychotherapy can be used to address some of the resulting problems of living with

ADHD, such as the effects of the disorder on self-esteem and family functioning (SIGN, 2001).

### **1.7.2. Pharmacological Treatments for ADHD**

It is estimated that up to 90% of children diagnosed with ADHD receive a medication trial at some point in their lives (Gadow, 1979). As such, the use of medication for the treatment of ADHD is a particularly well studied topic, generating more research than any other area of child and adolescent psychopharmacology (Coghill, 2003). Currently, there are three main types of medication used – psychostimulants, nonstimulant-noradrenergic reuptake blockers and (alpha)-agonist antihypertensive agents (DeNisco et al., 2005). Psychostimulants are the most commonly prescribed psychotropic agent for children. These typically come in the form of short-acting immediate release stimulants, such as methylphenidate. Methylphenidate is believed to work by affecting neurotransmitters within the frontostriatal regions of the brain. As mentioned, it has been suggested that people with ADHD may have an excess of dopamine transporters, thus resulting in low levels of dopamine within the brain. As dopamine is associated with motivation and reward (Himelstein et al., 2000; Volkow et al., 2001), the low levels found in ADHD may give rise to many of the symptoms which characterise the disorder e.g. poor response inhibition, impulsivity, inattention and hyperactivity. Methylphenidate works by influencing the processes involved in the uptake and release of dopamine, thus maintaining adequate levels of this neurotransmitter within the brain. By doing

so, methylphenidate allows interest in less motivating tasks to be maintained, and performance to be improved (Volkow et al., 2001).

Methylphenidate has a 1-4 hour duration and a half-life of 2-3 hours. Because of its short half-life, it is administered three times a day, eliciting concerns regarding compliance, privacy and stigmatisation (DeNisco et al., 2005). Such problems have led to the development of several long-acting formulations of psychostimulants, which are becoming increasingly popular with clinicians, patients and their families. These include a number of once-a-day preparations of methylphenidate (Concerta, Metadate CD and Ritalin LA) which act by:

*“carrying the active stimulant molecule through a slow-release biphasic delivery system”* (Denisco et al., 2005, pg. 17).

Indeed, there is evidence to suggest that stimulant medication significantly improves both the classroom and social behaviour of children with ADHD (Pelham, 1986). In a recent review of eighteen studies, Miller et al. (1998) found that, in comparison to placebo, methylphenidate, dextroamphetamine and pemoline treatments all resulted in significantly superior teacher ratings of behaviour.

Contentions of improved functioning are also supported by the neuropsychological evidence. In comparison to unmedicated ADHD controls, children receiving stimulant treatment demonstrate enhanced performance on tests of executive

functioning, including spatial working memory, set-shifting and planning (Kempton et al., 1999). This is in contrast to animals and human controls, whose executive functioning skills are impaired by such drugs (Dyme et al., 1982). Such psychotropics can also improve attentional processes. For instance, a recent study by Hood et al. (2005) found that, following administration of methylphenidate, children with ADHD demonstrated significant improvements in the Test of Everyday Attention – a neuropsychological battery designed to assess various aspects of cognitive attention.

### **1.7.3. The Effects of Medication on Response Inhibition**

With stimulant medication being recommended as the first-line treatment in ADHD (NICE, 2000), and the dominant neuropsychological theory citing response inhibition as the cardinal feature in ADHD (Barkley, 1997), it is perhaps unsurprising that recent research has begun to look at the relationship between these factors.

A recent study used the Go/NoGo task, alongside a number of electrophysiological measures, in order to examine the effects of methylphenidate on response inhibition in children with ADHD (Broyd et al., 2005). In terms of task performance, the ADHD group were found to make more overall errors (omission and commission) in the pre-medication condition. After receiving medication, the ADHD group continued to make more omission errors than controls, but no longer differed from controls with regard to the number of commission errors. The authors interpreted this as evidence that methylphenidate does ameliorate deficits in response inhibition

and these behavioural findings were further substantiated by the electrophysiological findings of reduced skin conductance levels, overall supporting a hypoarousal model of ADHD (Broyd et al., 2005).

Scheres et al. (2003) investigated the effect of methylphenidate and placebo on response inhibition in 23 boys with ADHD. However, in this study, response inhibition was broken down and assessed specifically in relation to the proposed three components – i.e. inhibition of a prepotent response, inhibition of an ongoing response, and interference control. Inhibition of a prepotent response was measured using two versions of the Stop Paradigm. A Circle Tracing Task and a Follow Task was used to assess inhibition of an ongoing response, while both the Stroop Colour-Word Test and an Eriksen Flanker Task (where subjects have to suppress an automatic reading response to irrelevant stimuli, adjoining the target) were used to measure interference control. Results revealed that, compared to placebo, inhibitory control did improve with methylphenidate. However, this effect was only significant for inhibition of a prepotent response and the Follow Task measure of inhibition of an ongoing response. In contrast, there was no significant effect of methylphenidate on interference control. This is in contrast to the findings of a previous study where, in comparison to pre-treatment baseline assessment, Stroop performance was reported to have improved following one year of methylphenidate treatment (Everett et al., 1991). Although the study also investigated the effects of methylphenidate dose by employing four treatment conditions: placebo, 5mg MPH, 10mg MPH,

20mg methylphenidate, there were no effects of medication dosage on any of the response inhibition measures.

Overall then, pharmacological techniques have generally been found to improve both the behavioural and neuropsychological performance of children with ADHD. It should be noted, however, that concerns have been raised with regard to the use of psychostimulant medication for children, particularly young children, with ADHD. Perring (1997) highlights ethical issues pertaining to the use of medication to modify children's behaviour. Pelham et al. (1998) go on to raise concerns about the limited, short-term effectiveness of these stimulants and others have expressed disquiet about the lack of information regarding the potential long-term side-effects that these drugs may have on children's physical and neurological development (Sonuga-Barke et al., 2003).

Given these concerns and an on-going question over the best long-term treatment for ADHD, the US National Institute of Mental Health sponsored an ongoing, multi-site treatment study of children with ADHD – i.e. The Multimodal Treatment Study of Children with ADHD (MTA).

#### **1.7.4. Combination Therapy and the MTA Study**

Given pharmacotherapy is considered efficacious as a stand-alone treatment (e.g. Klassen et al., 1999) it might be expected that a combination of drug therapy alongside another type of therapy would be as at least (if not more) effective.



However, results appear to offer only minimal support for this expectation. For instance, reviewing three studies which measured outcome using behavioural ratings, Miller et al. (1998) concluded that combined treatments did not differ significantly from drug treatment alone. Similar results were found in four out of the five studies reviewed by Jadad et al. (1999).

To address these issues in more detail, the US National Institute of Mental Health conducted a large-scale Multimodal Treatment Study of Children with ADHD (MTA Cooperative Group, 1999). The study involved a total of 579 children aged seven to nine years who were allocated to one of four treatment divisions and assessed over 14 months. The study compared medication management, behavioural treatment, combined treatment and standard community care. Results showed that combined and medication-only therapies were superior to behavioural treatments and community care with regard to reducing the symptoms of ADHD.

Although, the MTA is potentially the most rigorous study of its kind to date, it has been the subject of much criticism. Rubia & Smith (2001) argue that the

***“positive outcome for medication was predetermined by the study design”*** (Rubia & Smith, 2001, pg 313).

Indeed, on closer examination, many aspects of the study do seem biased in favour of the medication management condition. For instance, both the quality of care and





drug dosages were considerably higher in the medication management condition in comparison with standard community care (Jensen et al., 1999). Greene and Ablon (2001) also discuss the imbalance between the extents to which treatments were individually tailored. While drug treatment was comprehensively tailored to meet each child's individual needs, there was very little difference in the treatment regime delivered to children in the psychosocial intervention group. More significantly, no theoretical justification was provided for the strategies incorporated within the behavioural intervention leading some authors to query whether aspects of the programme were actually counterproductive (Morrell & Murray, 2003). Even if theoretically sound, the utility of the psychosocial intervention appears dubious, with claims that it would be impossible to replicate in more natural, clinical settings (Greene & Ablon, 2001). Furthermore, while those children in the medication management condition were actively receiving treatment at the time of outcome measurement, the level of input in the psychosocial condition had, by that stage, been tapered considerably (Jensen et al., 1999).

More thorough inspection of the data revealed a number of effects which have a considerable impact on the initial interpretation of results. Firstly, children in the medication management condition received higher doses of medication than those in the combined treatment (Jensen et al., 1999). Thus, combined treatment produced comparable outcomes using lower drug dosages, raising a question over whether the combined treatment approach would have emerged superior had drug doses been kept equal (Rubia & Smith, 2001). Although behavioural treatment was found not to

differ from standard community care on the three core symptoms of ADHD, it was found to be as good as medication management on almost all of the 16 associated ADHD symptoms, such as parent and teacher rated social-skills, relationships and academic achievement (Jensen et al., 1999). Concerns have also been raised over the potentially deleterious effect of the combined group having received medication *before* beginning behavioural treatment (Rubia & Smith, 2001). Finally, like many of the major studies and reviews regarding treatment in ADHD, the study failed to determine whether results were applicable to an older adolescent and/or adult population (Rubia & Smith, 2001).

Nonetheless, based on the originally reported findings that psychosocial intervention made no significant improvements on outcome when combined with medical treatment (MTA Cooperative Group, 1999) the British National Institute of Clinical Excellence report (NICE, 2000) recommended that medication should be used as the frontline intervention for ADHD, only followed by psychosocial interventions if necessary. It was only after this document was published that subsequent analysis, employing different outcome measures of ADHD symptoms, revealed that combined intervention was in fact superior to medical management alone when considering the long-term effects of treatment (Swanson et al., 2001).

The original study continues to dominate a lot of the current thinking and further misinterpretations by media and academia have fuelled contentions that medication alone is the most effective treatment for ADHD (Rubia & Smith, 2001). Thus,

despite several challenges to these findings (e.g. Daley, 2006; Owens & Hoza, 2003; Rubia & Smith, 2001), well-titrated medical treatment remains the treatment of choice for children with ADHD (Hood et al., 2005). Nonetheless, there are remaining concerns regarding the limited short-term effectiveness of medication (Pelham et al., 1998), as well as the potential long-term implications that taking these drugs may have on both the child's physical development (Sonuga-Barke et al., 2003) and their self-perceptions (Bugental et al., 1977).

### **1.8. Behavioural Attributions and Locus of Control in Children with ADHD**

Behavioural Attributions and Locus of Control in Children with ADHD is an area attracting growing interest. The focus on attributional effects is based on a social-cognitive framework within which causal attributions that one offers for others' behaviour are seen as being influential in guiding reactions to that behaviour (e.g. Weiner, 1993). For instance, parent attributions have been found to determine parenting responses to children (Bugental & Johnston, 2000), with parents being more likely to respond negatively to misbehaviour when they view the child as being responsible for their actions (Slep & O'Leary, 1998).

Attributions play an important role not only in guiding our interactions with others, but also in our own self-perceptions. Attributions for one's own behaviour are viewed as influential in predicting adaptiveness of functioning (e.g. Dweck et al., 1995; Taylor & Brown, 1988). For instance, an attributional style of seeing success

as due to external factors might be considered consistent with a helpless or depressogenic attributional style (see Milich, 1994).

Locus of control has been defined as the awareness of an association between one's actions and their consequences (Rotter, 1966). People who believe that an outcome is largely contingent upon their own behaviour are seen as having a more internal locus of control. Conversely, those with a more external locus of control tend to believe that outcomes are largely determined by other factors such as luck, fate, chance or powerful others. Locus of control has been shown to be associated with a number of different factors, including academic achievement (Nowicki & Kalechstein, 1997), psychological well-being and beliefs (see Lefcourt, 1983). Locus of control appears to be an important factor in the choices people make. For instance, a child with an internal locus of control, who perceives a connection between his or her behaviour, trying hard at school, and outcome/academic achievement, may be more likely to try hard at school (Nowicki & Kalechstein, 1997).

Research using measures of locus of control have shown that, in comparison to control subjects, children with ADHD have significantly higher external locus of control and are consequently more likely to view their experiences as being outwith their personal control or due to other, external factors (Hoza et al., 1993; Lufi & Parish-Plass, 1995).

This may be of particular concern when we consider that having accurate control beliefs and positive self-concept have been identified as important coping resources in relation to a child's ability to manage stress (Beitchman et al., 1992). Given the various aforementioned stressors associated with ADHD, it would seem important to examine such characteristics. Also, in view of the fact that successful academic performance primarily relies on learning new and challenging material, a child's response to difficult tasks may influence their ultimate success as learners (Milich, 1994). However, despite the clear importance of such factors, the majority of research focuses on the medical or biological aspects of ADHD, rather than the personality of the child (Lufi & Parish-Plass, 1995).

#### **1.8.1. Medication and Behavioural Attributions**

Some research has attempted to marry both aspects by examining behavioural attributions in children with ADHD and addressing the potential effect that medication may have on these processes. This stemmed from several authors (e.g. Bugental et al., 1977) expressing concern that successful treatment with medication may have an adverse effect on children's self-perceptions - arguing that it may be leading them to attribute their behaviour to external factors (e.g. the drug) while viewing their own efforts/abilities as having a relatively minor role. It has been speculated that this may result in children becoming reliant on drugs to focus their attention and effort, meaning that when medication is discontinued, they are left feeling that they have no way of controlling their behaviour (Rosen et al., 1985). As such, any potential effect that medication may have on the attributions made, both by

the child themselves and by others, may have a crucial influence on the child's overall development.

The existing evidence in this field is varied. Some case studies (e.g. Rosen et al., 1985) and indirect evidence suggests that medication does lead children to attribute their behaviour to more external factors. For instance, children with ADHD have been found to refer to their problems on a physiological basis and report that taking their medication does help them to control their behaviour (Henker & Whalen, 1980). In addition, a more recent study by Johnston & Leung (2001) involved boys with ADHD and their parents watching videos of child ADHD symptoms. Whilst there were few treatment effects in the attribution ratings of the child participants, it was noted that both parents and children attributed greater control and intent for negative behaviours during the medication condition. As such, the authors remark that these attributions:

***“...might generally be considered a maladaptive attributional style with negative ramifications for adaptive functioning.”*** (Johnston & Leung (2001), pg 74).

However, these studies have looked at behaviour in more general terms rather than in the context of performing specific tasks. Furthermore, the latter study used footage of unfamiliar children, making it unclear whether these results can be generalised to children's perceptions of their own behaviour.

In addressing such shortcomings, several studies have utilised specific academic tasks to examine the effects of stimulant medication on children's attributions for

their performance. For example, Milich et al. (1989) assessed a group of boys with ADHD on a test of sustained attention – The Continuous Performance Task - during both ‘medication’ and ‘placebo’ conditions. Despite the fact that the boys performed significantly better on medication compared to placebo, there were no significant differences in participants’ mean self-evaluations and attribution responses across these conditions. In fact, finding an increased correspondence between performance and self-evaluations within the medication condition, the authors suggest that medication may enhance children’s ability/motivation to monitor their own behaviour, leading to better task strategies.

Extending this work, research has compared boys with ADHD’s response to failure under medication and placebo conditions (Milich et al., 1991; Carlson et al., 1993). Participants were asked to complete a series of word-finding puzzles where success and failure were manipulated by exposure to solvable and unsolvable puzzles. Milich et al. (1991) found that, following the failure experience, boys made significantly more external attributions and significantly fewer internal attributions on medication relative to placebo. As such, the use of medication in the unsolvable condition appeared to reduce their perceived sense of responsibility for failure. On the other hand, Carlson and colleagues found that boys with ADHD attributed success to their efforts and failure to task difficulty regardless of medication status (Carlson et al., 1993). Furthermore, both studies found that, in comparison with the placebo condition, participants persisted with the unsolvable puzzles to a greater degree whilst on medication. Thus, reviewing the results of both studies together,

Milich (1994) suggests that medication may somehow ameliorate failure experiences, while simultaneously facilitating a healthier and more adaptive style of responding.

In general then, despite some inconsistencies in results, studies using academic-type tasks tend to conclude that there is no evidence to suggest that medication has a detrimental effect on children's attributions for their behaviour. Instead these children typically attribute success to effort and failure to external factors such as task difficulty (Johnston & Leung, 2001). What's more, findings of increased external locus of control in children with ADHD appear to be consistent irrespective of medication status (Lufi & Parish-Plass, 1995).

Nonetheless, it seems clear that, in children with ADHD, the effect medication can have - both on their behaviour directly and the related attributions made for such behaviour - is hugely important to their overall development. Generally, most of the studies to date which examine the effects of medication alongside attributional responses in children with ADHD, measure task performance using tests of sustained attention and/or assess their social/behavioural conduct. However, there seems to be limited research looking at the direct effects of medication on response inhibition or the influence that medication may have on the attributions for performance in such abilities. Considering response inhibition is now widely viewed as the cardinal feature of ADHD (Barkley, 1997), this seems an important area for future research to address.



## **1.9. Summary**

ADHD is a condition that has a significant impact on the lives of individuals and their families. Individuals with ADHD have been shown to have impairments in executive functioning (Pennington & Ozonoff, 1996) and findings of deficits in neuropsychological tests of response inhibition, support the now dominant theory citing response inhibition as the central deficit of ADHD (Barkley, 1997). Current recommendations advocate stimulant medication as the first-line treatment for ADHD (NICE, 2000). Concerns have been raised about the potential for medication to have an adverse effect on children's self-perceptions - leading them to attribute their behaviour to external factors, such as the drug, while viewing their own efforts/abilities as having a relatively minor role (Bugental et al., 1977). This may be of particular concern when we consider that having accurate control beliefs and a positive self-concept have been identified as important coping resources in relation to a child's ability to manage stress (Beitchman et al., 1992). Furthermore, successful academic performance primarily relies on learning new and challenging material, thus a child's response to difficult tasks may influence their ultimate success as learners (Milich, 1994). There is currently very little research looking at the direct effects of medication on response inhibition or the influence that medication may have on the attributions for performance in such abilities. Given that response inhibition is now widely viewed as the cardinal feature of ADHD (Barkley, 1997), this seems an important area for research to address.

### **1.10. Aims**

The aim of the current study is to examine whether methylphenidate is effective in improving response inhibition in children with ADHD. The study will employ a newly developed measure of response inhibition – the Animal Stroop Task (Wright et al., 2003). In addition, the study intends to examine children's own attributions about their level of response inhibition and will assess this factor across a variety of variables, including medication status.

### **1.11. Hypotheses**

1. In comparison with control participants, children with ADHD will show more impaired response inhibition, as measured by their 'reaction time difference' scores, on the Animal Stroop Task (Wright et al., 2003).
2. Following their prescribed methylphenidate dose, children with ADHD will show an improvement in response inhibition as measured by their 'reaction time difference' scores, on the Animal Stroop Task.
3. In comparison to control participants, children with ADHD will be significantly more likely to have an externally-based locus of control.

4. Children who have a more internally based locus of control will demonstrate better inhibitory control, as measured by their Reaction Time-Difference Score on the Animal Stroop.

5. Children with ADHD in the unmedicated condition will give lower estimations of both predicted- and post- performance ratings in comparison with their ratings in the medicated condition and with the ratings of control participants.

6. There will be a difference between the participant groups in the self-evaluation ratings of performance:

(a) In comparison to control participants, children with ADHD will be more likely to attribute their performance to external factors.

(b) Children with ADHD will be more likely to attribute their performance to external factors in the unmedicated condition in comparison to the medicated condition.

## **2. Method**

### **2.1 Design**

A between subjects design was used to investigate the performance of children with ADHD and a control group on measures of response inhibition, locus of control and behavioural attributions.

Additional within subjects comparisons were made in the group of participants with ADHD to compare response inhibition and behavioural attributions in the 'medicated' vs. 'unmedicated' condition.

#### **2.1.1 Power Calculation**

Due to the fairly recent release of the Animal Stroop Task as a measure of inhibitory control, there is currently a very limited amount of research utilising this test. Nonetheless, using Cohen's (1992) formula for calculating effect size (for tests of difference) a large effect size was posited from previous research articles of a similar nature (e.g. Lufi et al., 1990; Pennington & Ozonoff, 1996; Scheres et al., 2004). Based on Cohen's (1992) estimate of sample size (setting power at 0.8 and alpha at 0.05) one-tailed between subjects tests of difference would require that  $N=20$ , and one-tailed within subjects tests of difference would require that  $N=12$  (Clark-Carter, 2004).

## **2.2 Participants**

Two groups participated in this study, an experimental group of individuals with Attention Deficit Hyperactivity Disorder (ADHD) (n= 21) and a control group of non-ADHD individuals, (n= 25) matched for age, gender and IQ.

### **Experimental Group**

The children in the experimental group were current patients of the Child and Adolescent Mental Health Service (CAMHS) in the Scottish Borders. All participants in the experimental group had been given a diagnosis of ADHD by a consultant psychiatrist or multi-disciplinary team prior to taking part in the study and were currently being treated with psychostimulant medication.

### **Control Group**

Participants in the control group were recruited from a variety of local schools. They had had no previous contact with the Child and Adolescent Mental Health Service and did not have a diagnosis of ADHD.

#### **2.2.1 Inclusion Criteria**

To be included in the study, children in the experimental condition had to meet the following criteria:

1. Aged between 5-16 years
2. Primary diagnosis of ADHD
3. Be receiving treatment with psychostimulant medication

Children in the control group had to be:

1. Aged between 5-16 years
2. No diagnosis of ADHD

### **2.2.2 Exclusion Criteria**

1. Children who were diagnosed with any other significant mental health condition (e.g. autistic spectrum disorder) were excluded from the study.
2. Children with a learning disability (IQ below 70 and significant impairment in adaptive functioning) were excluded.
3. Children with ADHD who were prescribed non-methylphenidate based drugs (e.g. Atomoxetine) were also excluded.

### **2.2.3 Ethical Approval**

Ethical approval was granted by the Borders Research Ethics Committee (see appendix 7.2). A detailed discussion of the ethical issues that arose and were addressed in relation to the research methodology is given in section 2.5 below.

## **2.2.4 Recruitment**

### **Experimental Group**

Based on the above criteria, suitable participants for the experimental group were identified by members of a multi-disciplinary 'ADHD Team', as well as consultant psychiatrists within the larger CAMH Service. The parents/guardians of potential participants were contacted by letter and invited to participate (appendix 7.3). They were sent two, separate versions of the information sheet – one for themselves (appendix 7.4) and one, age-appropriate version for the child (appendix 7.5). Consent forms were also included for both parents/guardians and children (appendices 7.6 and 7.7) and parents/guardians were asked to specify whether they would prefer their child to be seen at school or at the child and adolescent unit. Parents/guardians were also informed that it may be possible for the researcher to visit their child at home and were asked to indicate this preference on the form. Once consent was received, parents/guardians were contacted by telephone and arrangements were made to see the child. Details of each child's prescribed psychostimulant medication were also gained at this point. All participants were being treated with psychostimulant medications – Concerta XL, Ritalin or Equasym – but the dosages received by each participant varied dependent on their individual requirements. The length of time that participants had been receiving psychostimulant medication varied from between 4 and 84 months (mean = 17.810, SD = 17.332).

## **Control Group**

The matching requirements of the control group were established after identifying participants in the experimental group. Teachers from five local schools were contacted regarding the study. They agreed to distribute the letters across the various year-groups required, inviting parents/guardians and their child to take part in the study (see appendix 7.3). The teachers were asked to select children who were considered to have no/few difficulties with hyperactivity or attention and who were not known to have mental health problems. It was recommended that they distribute invitations to children from a mixture of academic abilities but refrain from selecting those with significant learning difficulties. As with the experimental group, parents/guardians and their children each received separate copies of the information sheet and consent forms and parents/guardians were asked to indicate where they would prefer their child to be seen. (Appendices 7.4 – 7.7 contain copies of the information sheets and consent forms distributed to participants in the control group).

### **2.2.5 Response Rate**

## **ADHD Group**

Of the 98 families contacted, 25 consented to participate in the experimental group, a response rate of 25.51%. Of those 25, four were automatically excluded from the study due to incomplete data.



## **Control Group**

Of the 34 families contacted, 25 consented to take part in the control group, a response rate of 73.53%. All 25 control participants were included in the study.

## **2.3 Description and Application of Measures**

The following section describes the materials used to measure each variable and the manner in which these were applied.

### **2.3.1 Estimated Full Scale IQ**

Full-scale IQ was estimated using a shortened version of the Wechsler Intelligence Scale for Children, UK (WISC-III UK, 1991). The WISC-III is a wide-ranging assessment measure for use with children aged 6-17 years. It yields a full-scale IQ score by examining verbal and performance abilities, over 13 different subtests. It takes approximately two hours to complete and provides information regarding the child's relative skills in different areas compared to a large normative group of children of the same age and gender. The shortened version employed comprised one verbal subtest (Vocabulary) and one performance subtest (Block Design). These two subtests have high correlations with the Full Scale IQ and consistently high reliabilities (Sattler, 1992). Participants' scaled scores on these subtests were combined in order to estimate a Full-Scale IQ score based on the method described by Sattler (1992). The validity of this short form is high ( $r = 0.906$ ) (Sattler, 1992) and it is considered to be a good screening combination (Kilian & Hughes, 1978).

### 2.3.2 Response Inhibition

Response inhibition was measured using the Animal Stroop task (Wright et al., 2003). The Animal Stroop task is a relatively new, pictorial modification of the original Colour-Word Stroop Task (Stroop, 1935). The Animal Stroop task is based on four exemplar, animal stimuli - a cow, a pig, a duck and a sheep. The task comprises three conditions. The first is an 'Incongruent Condition' where each of the animals' heads is substituted with a head from another of the three animal prototypes. Thus, within the incongruent condition, there are 12 animal-stroop stimuli. The second condition is a 'Matching Condition' where each of the four animal prototypes is displayed as a whole animal - i.e. the animal's body is coupled with the appropriate, matching head. Lastly, the task includes a 'Control Condition' where the animal's head is replaced by a caricature of a face. The stimuli used in each of the three conditions are displayed in Figure II.




Trial Type	Stroop	Animal – Stroop
Stroop	RED	
Matching	RED	
Control	CHAIR	

Figure II. Stimuli used in standard stroop and animal-stroop task

The Animal Stroop task is based on the premise that facial information is preferentially processed (Johnson, 1993) and utilized preferentially in semantic categorization (Quinn & Eimas, 1996). Thus, in both the incongruent and control conditions, stroop-like interference can be induced by asking the child to name the animal's body and inhibit a preferred response to identify the head (Wright et al., 2003). The control task is intended to act as a semantic control in that it contains similar semantic content as a face, but produces less activation of animal representations. As such, it is believed to serve as the most appropriate comparison with the incongruent task (Wright et al., 2003).

The Animal Stroop task comprises three blocks, with twenty four images in each. The first and third blocks consist of a mixture of incongruent and control images – twelve of each within a block. The second block contains 'matching' images only. In blocks one and three, children are required to name the animal's body whereas, in block two, they are asked to simply name the animal. The difference between reaction times in the incongruent and control conditions (i.e. 'Reaction Time Difference') is used as a measure of inhibitory control (Wright et al., 2003). Thus, blocks one and three are used to provide a measure of response inhibition. The images are presented on a computer screen and displayed in a random order. Each picture is shown in one of two orientations – i.e. either with the animal's head to the left or to the right of the screen. A fixation point is displayed in the centre of the screen for 0.5 seconds. Following each fixation point, an animal image is presented for three seconds, during which time the child is required to name the image. After

three seconds, the image is removed from the screen and is followed by a one second interval prior to the presentation of the next fixation point.

Before commencing the Animal Stroop Task, participants were shown flashcards of each of the four animals and asked to name them in order to ensure correct identification. Following this, participants were given verbal task instructions and completed a series of 'warm-up' trials. An example image was presented at the start of each block whereupon the task instructions were repeated. Participants were required to identify the example image correctly before proceeding with the test (a copy of the task instructions appear in appendix 7.8).

The accuracy of children's naming was recorded by the experimenter (see appendix 7.9 for a copy of the scoring sheet). Reaction times were recorded by 'voice key', elicited by the participants' vocal response, with the researcher operating a manual timing procedure as back-up.

### **2.3.3 Locus of Control**

Locus of Control was measured using the Locus of Control Scale for Children (LCSC) (Nowicki & Strickland, 1973) - a brief 40-item pencil and paper questionnaire. The measure is suitable for use with children aged 9 – 18 years old and asks questions about the extent to which behaviours and their contingencies are seen as under the control of self. People who tend to view such matters as being within their control are more likely to have an 'internal locus of control'.

Conversely, if the consequences of behaviour are attributed to luck, chance or the actions of powerful other means, an 'external locus of control' is present. Each item is rated either 'yes' or 'no' and the total is used to provide a single, dimensional score of the degree of external/internal locus of control. The scale takes approximately 10 minutes to complete and has relatively good reliability and validity (Nowicki & Strickland, 1973). For children under the age of nine, a twenty-six item, pictorial version of the scale – the Preschool and Primary Nowicki-Strickland Internal-External Scale (PPNSIE) (Nowicki & Marshall, 1974) – was used. The PPNSIE allows for a parallel, age-appropriate reliable and valid measure of locus of control for children aged 4-8 years old (Nowicki & Marshall, 1974). All questions were read aloud by the researcher and participants' responses recorded. On some occasions where the child did not understand the question, further verbal information was provided.

In order to compare the two forms of the test, the percentage of external items was calculated for each participant. This is recognised as being the primary method of comparing the two scales (Nowicki, personal contact, 2006).

#### **2.3.4 Self Evaluation and Attribution Questionnaire**

Because Locus of Control Scales are often used to measure more general expectancy and are mainly concerned with the expectation of future events, it has been argued that they should not be used to predict actions in specific situations or activities (Furnham & Steele, 1993). On the other hand, attributional measures are more concerned with the causes of past events (Furnham & Steele, 1993), thus a more

focused attribution questionnaire was used to assess participants' experience of the animal stroop task. Due to a lack of standardised measures relating to the specifics of the tasks employed, the researcher developed this short questionnaire based on previous research examining attributions (Milich et al., 1989).

Because children's attributions vary depending on whether they are describing successes or failures (Bar-Tal & Darom, 1979), participants were asked whether they felt they would/had done a good or a bad job before and after completing the animal stroop task. Participants were also asked to predict how well they thought they would perform on the task. The questionnaire then went on to assess a number of dimensions based on questions used in a previous study examining attributions by Milich et al. (1989). Using a 10-point scale, participants were asked to rate (a) how well they thought they had performed on the task; (b) how easy they thought it had been to pay attention; (c) how hard they felt they had tried; (d) how lucky they felt they had been; (e) how hard they thought the task was. Participants in the experimental group were also required to rate an additional question: (f) how much they felt their medication had helped them on the task. Finally, participants were asked to rank the above factors in order from most to least important. Due to the extra question used with the experimental group, participants with ADHD were asked to rank the importance of five factors – 'attention'; 'trying'; 'luck'; 'task difficulty' and 'medicine'. Accordingly, children in the control group were asked to rank the importance of only four factors – i.e. attention, trying, luck and task difficulty. A copy of this questionnaire is contained in appendix 7.10.

### **2.3.5 Strengths and Difficulties Questionnaire (SDQ)**

Although the school teachers had been asked to select control children who they considered as having few/no difficulties with attention or hyperactivity, the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997) was used as a more formal measure of assessing levels of hyperactivity in control group participants.

The SDQ is a brief behavioural screening tool used to assess children between the ages of 3 – 16 years. It comes in a number of informant and self-rated versions. The questionnaire examines attributes across five different scales – emotional symptoms, conduct problems, hyperactivity/inattention, peer relationship problems and prosocial behaviour – each containing five items. Respondents are asked to rate each question as ‘Not True’, ‘Somewhat True’ or ‘Certainly True’. A total difficulties score (ranging from 0-40) is generated by summing the scores from all the scales except the prosocial scale. Scores can be classified as ‘normal’; ‘borderline’ or ‘abnormal’ using bandings outlined by the authors. These bandings were derived with reference to normative data and differ depending on the type of informant who completed the questionnaire (i.e. parent or teacher). The reliability and validity of the SDQ are relatively good and, as such, it is considered as a useful, brief measure of the adjustment and psychopathology of children and adolescents (Goodman, 2001).

A strengths and difficulties questionnaire was completed for each participant in the control group - either by their parent(s) or classteacher. All participants in the

control group scored within the ‘normal’ range with regard to ‘hyperactivity/inattention’.

## **2.4 Procedure**

All participants were tested on a one-to-one basis, in a separate room, either at school, at the Child and Adolescent Unit or at home. In the minority of cases where another adult was present, they did not contribute to the assessment. In order to ensure consistency of the testing procedure, all participants were assessed by the same researcher.

### **Experimental Group**

Participants in the experimental group were seen on two separate occasions. Each participant was assessed under two conditions – a ‘medicated’ condition where they had taken their prescribed stimulant medication as usual, and an ‘unmedicated’ condition where they were assessed following an unmedicated period of at least 12 hours. Arrangements for the day of the ‘unmedicated’ assessment session were agreed with parents by telephone and participants prior to meeting with them and each parent was sent a letter confirming these plans (see appendix 7.11 for an example). The majority of children were assessed first thing in the day, allowing participants and their parents to have the option of simply delaying their morning dose of medication – i.e. participants were able to take that day’s dose of medication by 10am – approximately only two hours later than what would typically have been the case. For those receiving short-acting psychostimulant drugs administered



two/three times a day, each subsequent dose was also postponed a further two hours. In some cases, parents opted for their child to miss their morning/once-daily dose of medication altogether. In all circumstances, participants and their parents were informed that the child should return to their usual medical regime the following day. The researcher contacted each child's school directly to notify them of these arrangements and respond to any questions or concerns that they had. The GPs of all participants in the experimental group were also notified by letter (see appendix 7.12) and given the chance to contact the department if they had any queries.

Equal numbers of participants were allocated to the 'medicated' or 'unmedicated' conditions initially and the condition-type was reversed for their second assessment session. The period of time between assessment sessions varied for each participant and ranged from between four and fourteen days (mean = 6.810, SD = 2.040).

In the 'medicated' condition, participants completed the aforementioned measures in the order presented below:

- a) Question 1 of the Attribution Measure – asking the child to predict their performance on the Animal Stroop task.
- b) The Animal Stroop Task
- c) The remainder of the Attribution Questionnaire
- d) The Wechsler Intelligence Scale for Children – Version III (WISC-III) – shortened version.

- e) The Locus of Control Scale for Children (LCSC) (for participants aged 9 years and over) / Preschool and Primary Nowicki-Strickland Internal-External Scale (PPNSIE) (for participants aged 8 years and below).

The full testing battery took between 40 and 60 minutes to complete depending on the participant.

In the 'Unmedicated Condition', participants completed sections a-c. In this session, assessment time varied between 15 and 30 minutes.

### **Control Group**

Participants in the control group were seen on only one occasion and completed the measures as outlined in the above 'Medicated Condition'. During this session, a Strengths and Difficulties Questionnaire was completed by either the child's parent or classteacher. As above, assessment time varied between 40-60 minutes for each of the control participants.

Before commencing each of the measures, all participants received verbal task instructions and the researcher verified each child's understanding before proceeding with the test. All participants were informed that they could discontinue at any time.

All results of the tests and questionnaires were subsequently scored and recorded on an SPSS database and statistically analysed using SPSS (Statistical Package for the Social Sciences) for Windows, Version 11.

## **2.5 Ethical Considerations**

Adhering to the British Psychological Society (BPS) Good Practice Guidelines for the Conduct of Psychological Research within the NHS (Cooper et al., 1993), appropriate steps were taken to ensure that the study was ethical and did not cause harm. As the research was investigating aspects of ADHD, a disorder which is primarily reported to affect children, the participation of a child sample was deemed necessary. However, given that children are classed as a vulnerable population, by referring to guidance notes on research involving children (University College of London's Research Ethics Committee, 2006), special consideration was given to including these participants in the study. Participants and their parents/guardians were provided with detailed, developmentally-appropriate information sheets (see appendix 7.4 – 7.5) and also given several opportunities to ask questions, in order that they could make an informed decision as to whether or not to fully consent to the research. While all participants assented to participate, given they were all under the age of sixteen, parental consent was also obtained.

The study method was designed to be appropriate for children and the circumstances in which the research was conducted provided for the physical, emotional and psychological safety of the child (University College of London's Research Ethics

Committee, 2006). As such, parents and children were offered a choice of venue and parents were also offered the option of being present during testing. Where parents were not present, the researcher ensured that a contact telephone number was available in case there were any difficulties. Given the dependent nature of children, there is a possibility that power differentials may characterize the relationship between child participants and the researcher. As such, both parents and children were made aware that they could withdraw from the study at any time and children were reminded of this option throughout. In view of children's reduced capacity for full understanding, the assessment procedure was explained to participants in detailed, developmentally-appropriate language and their understanding confirmed before commencing with the different measures. Each child was informed that the test items varied in difficulty and that they should not worry if some were too difficult as all people got stuck on parts. The researcher encouraged participants throughout the testing procedure. All participants were thanked for their participation, both verbally and by letter (see appendix 7.13). Participants, parents and schoolteachers were given the opportunity to ask questions and were asked if they wished to receive a summary of the completed study's findings. The participants were informed that all data would be made anonymous and confidentiality protected.

In addition, like all proposals involving research with children, the project was submitted to a research ethics committee and was granted approval (see appendix 7.2).

Great consideration was also given to the issue of assessing participants in the Experimental Group 'off medication'. In consultation with the Consultant Psychiatrist attached to the Borders CAMHS and ADHD Service, it was agreed that participants would only be required to miss a single dosage of their routinely prescribed medication prior to only one of two assessment sessions. This process was considered unlikely to have any significant adverse effects as, due to the relatively short half-life of stimulant medications, children prescribed these treatments often experience a dip in medication level during some points of the day (Pelham et al., 2001). For example, for each four hours of short-acting methylphenidate treatment, the medication is likely to be having an effect for only 2-3 hours. Although the slower release methylphenidate preparations, such as Concerta, are designed to last 12 hours, children will typically have commenced on a period of treatment with a shorter-acting stimulant preparation before being changed over to their longer-acting prescription. In addition, given the link between parents with a chaotic parenting style and children with ADHD (Johnston & Mash, 2001), it is not uncommon for children with ADHD to have unintentionally forgotten to take their medication at times. As such, children are accustomed to feeling 'medication-free', while parents and teachers are in the habit of managing any associated behaviours. The researcher was in close consultation throughout the study with the Child and Adolescent Psychiatrist and parents and children were encouraged to contact either the researcher or the Psychiatrist if they had any queries.

The researcher went through the required procedure in depth with each participant's parent(s). It was arranged for the majority of children to be assessed first thing in the day, allowing participants and their parents to have the option of simply delaying their morning dose of medication – i.e. participants were able to take that day's dose of medication by 10am – approximately only two hours later than what would typically have been the case. For those receiving short-acting psychostimulant drugs administered two/three times a day, each subsequent dose was also postponed a further two hours. In some cases, parents opted for their child to miss their morning/once-daily dose of medication altogether. Arrangements for the day of the 'unmedicated' assessment session were agreed with parents by telephone and participants prior to meeting with them and each parent was sent a letter confirming these plans (see appendix 7.11 for an example). In all circumstances, participants and their parents were informed that the child should return to their usual medical regime the following day. Where children were attending school on the day of their 'unmedicated' session, the researcher contacted the school directly to notify them of these arrangements and respond to any questions or concerns that they had. The GPs of all participants in the experimental group were notified by letter (see appendix 7.12) and given the chance to contact the department if they had any queries.

### **3. Results**

The first part of the results section is comprised of descriptive statistics. An outline of how the data was prepared for analysis will be presented initially. This will be followed by demographic information about the participants and a description of subsequent exploratory analyses. The second part of the results section details how each hypotheses was tested individually using inferential statistics.

#### **Terminology**

Inhibitory Control was measured by the Reaction-Time Difference Score of the Animal Stroop task, therefore the terms ‘Inhibitory Control’ and ‘Reaction Time-Difference’ are used interchangeably.

#### **3.1 Preparation of the data for analysis**

The distribution of the variables was investigated by examining the histograms, skewness and kurtosis scores for each variable (see appendix 7.14). The age and IQ variables were normally distributed. The other variables – the inhibitory control score (RT-difference), locus of control score and the individual responses on the attribution questionnaire - were examined for each group. The data for the reaction time difference scores and locus of control scores was normally distributed. However, the data for the remaining variables was not normally distributed in either of the groups. Approximately half of the variables were negatively skewed, while

the others were positively skewed. In order to meet requirements for parametric testing a natural logarithm ( $x+1$ ) transformation was therefore carried out on each variable. Transforming this data resulted in normal distributions.

The data was subsequently analysed using parametric tests with SPSS (Statistical Package for the Social Sciences) Version 11. Parametric tests were considered optimal due to contentions that they are more powerful (Dancey & Reidy, 2004) and robust (Clark-Carter, 2004) and, as such, may be less likely to commit Type II errors (Clark-Carter, 2004). The significance level of test results, unless otherwise stated, was set at  $p=0.05$  (one-tailed).

## **3.2 Descriptive Statistics**

### **3.2.1 Participant Demographics**

#### **Experimental Group**

The age of participants with ADHD ranged from 6-14 (mean CA = 9.33, SD = 2.58). A total of 16 boys and 5 girls participated. Estimated Full-Scale IQ (FSIQ) scores ranged from 62 - 144 (mean FSIQ = 94.29, SD = 18.16).

#### **Control Group**

Participants in the control group were matched with those in the experimental group for age, gender and estimated FSIQ. Twenty boys and 5 girls took part, aged



between 6 – 13 years (mean CA = 9.2, SD = 2.0). Estimated Full-Scale IQ scores ranged from 71 - 132 (mean FSIQ = 102.48, SD = 17.03).

Using independent samples t-tests, the two groups were statistically compared on age and IQ. No significant differences were detected. A chi-square showed there was no significant difference between the distribution of males and females in the two groups.

Table 1 (below) summarises the results of the tests of difference / association, for the experimental and control groups.

	ADHD Group (n = 21) X (SD) Range	Control Group (n = 25) X (SD) Range	Test of difference / association	P (2-tailed)
CA Chronological age	9.33 (2.58) 6-14	9.2 (2.0) 6-13	t = -0.20	0.84
WISC-III Estimated FSIQ	94.29 (18.16) 62 - 144	102.48 (17.03) 71 - 132	t = 1.58	0.12
Gender M:F	16:5	4:1	$\chi$ = 0.10	0.78

Table 1: Descriptive characteristics of the sample

### 3.2.2 Exploratory Data Analysis

Two participants were removed from each of the ADHD group (medicated condition) and the Control group due to outlier scores on the inhibitory control measure of the Animal Stroop. These scores needed to be removed in order for the groups to be matched on the inhibitory control task. Figure III (below) illustrates a box-plot of the inhibitory control task scores for the experimental (medicated condition) and control conditions. There were no outliers in the ADHD-unmedicated condition.

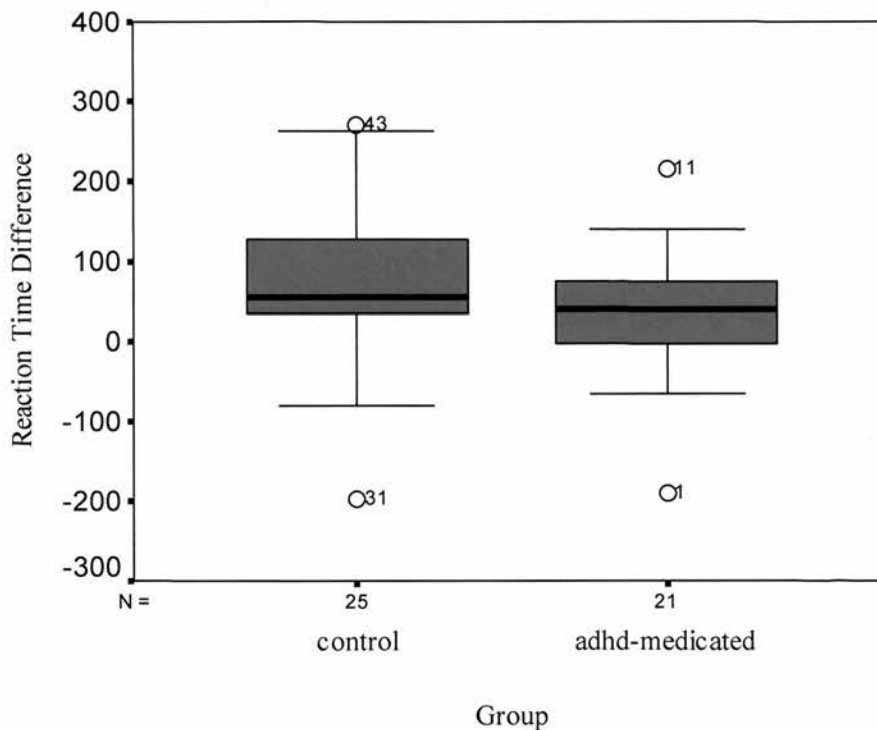


Figure III. A box-plot to show the scores on the inhibitory control task for the experimental (medicated condition) and control groups.

An ANOVA was used to examine whether there was any effect of the order in which the experimental participants were tested (i.e. whether they were in the medicated followed by unmedicated condition or vice versa). The ANOVA showed there was no interaction between order and group ( $F = 0.83$ ,  $p = 0.37$ ,  $df = 1$ ) – i.e. there was no combined effect of the two factors (order and group) on Reaction Time Difference scores (see appendix 7.15). The ‘order’ variable was therefore removed from further analysis.

### 3.3 Hypothesis Testing

Each hypothesis was tested using inferential statistics. Independent- and paired-samples t-tests were used to investigate hypotheses 1, 2, 3, 5 and 6. Pearson’s correlations were used to investigate hypothesis 4. The results for each hypothesis are reported below.

#### Hypothesis 1

**In comparison with control participants, children with ADHD will show more impaired response inhibition, as measured by their ‘reaction time difference’ scores, on the Animal Stroop Task (Wright et al., 2003).**

In order to investigate hypothesis one, two separate comparisons were made between: a) Participants with ADHD in the medicated condition (ADHD-medicated) and Control participants.

b) Participants with ADHD in the unmedicated condition (ADHD-unmedicated) and Control participants.

Independent Samples t-tests showed no significant differences in Reaction Time Difference Scores between either the ADHD-medicated and control groups ( $t = -0.98, p = 0.17, df = 44$ ) or between the ADHD-unmedicated and control groups ( $t = 1.56, p = 0.06, df = 40$ ). See table 4 and figure IV for a comparison of mean reaction time difference scores.

The null hypothesis cannot, therefore, be rejected.

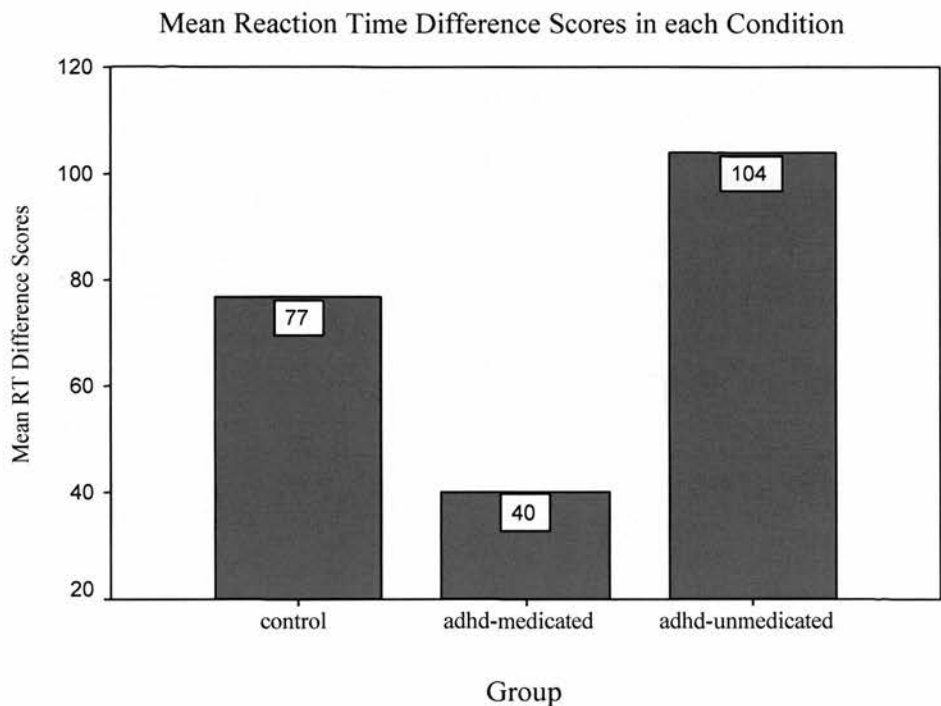


Figure IV. Reaction Time Difference Scores for both the Experimental and Control Groups.

	ADHD-Medicated X (SD) Range (n)	ADHD-Unmedicated X (SD) Range (n)	Controls X (SD) Range (n)
RT Difference	39.95 (58.60) -66.00 – 141.00 (19)	103.86 (103.02) -66.00 – 298.00 (21)	76.57 (86.99) -80.00 – 262.00 (23)

Table 4. Means, Ranges and Standard Deviations for Hypotheses 1 & 2.

## **Hypothesis 2**

**Following their prescribed methylphenidate dose, children with ADHD will show an improvement in response inhibition as measured by their ‘reaction time difference’ scores, on the Animal Stroop Task.**

A Paired-Samples t-test showed a significant difference between the reaction time difference scores of participants with ADHD-medicated and participants with ADHD-unmedicated ( $t = 2.28$ ,  $p = 0.02$ ,  $df = 18$ ). ADHD participants showed significantly higher reaction time difference scores in the unmedicated condition compared to the medicated condition. See table 4 and figure IV for a comparison of mean reaction time difference scores.

Hypothesis 2 is upheld and the null hypothesis is rejected.

### Hypothesis 3

**In comparison to control participants, children with ADHD will be significantly more likely to have an externally-based locus of control.**

An independent samples t-test showed there was no significant difference between the locus of control scores of experimental participants and participants in the control group ( $t = -0.77$ ,  $p = 0.22$ ,  $df = 44$ ). See figure V for a comparison of the mean percentage of externality scores across the two groups. Comparisons were made using the mean percentage of external items yielded by each participant group.

In the case of hypothesis 3, the null hypothesis cannot be rejected.

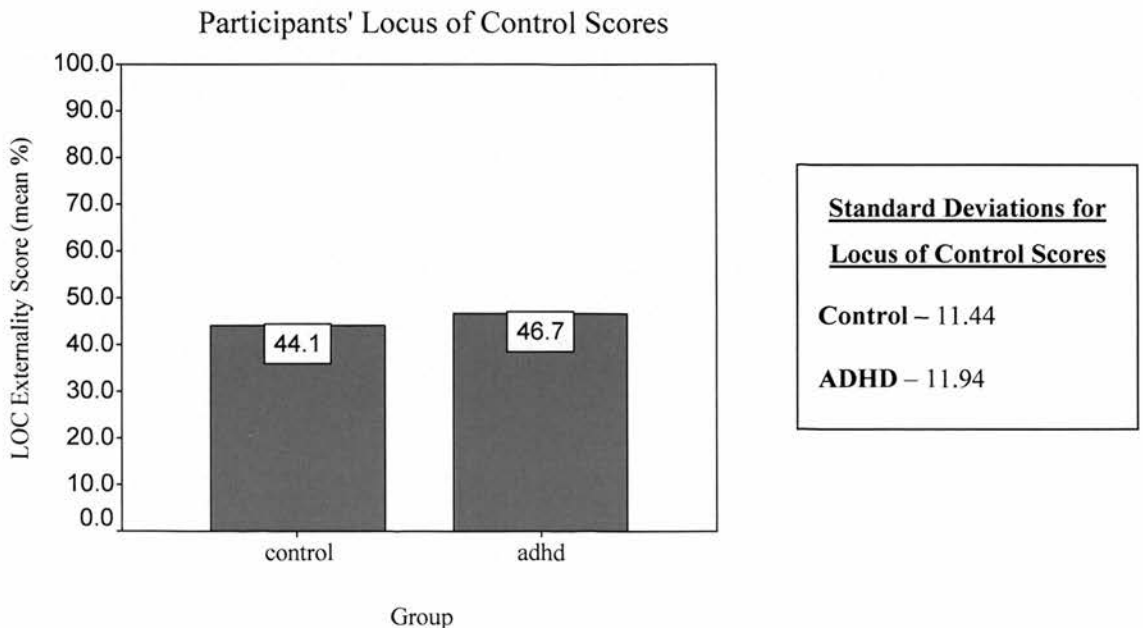


Figure V. A Comparison of External Locus of Control Scores across Participant Groups

#### **Hypothesis 4**

**Children who have a more internally based locus of control will demonstrate better inhibitory control, as measured by their Reaction Time-Difference Score on the Animal Stroop.**

Pearson's Correlations detected no significant relationships between Locus of Control scores and reaction time difference scores in any of the three conditions – i.e. ADHD-medicated ( $r = -0.08$ ,  $p = 0.38$ ,  $N = 19$ ); ADHD-unmedicated ( $r = 0.26$ ,  $p = 0.13$ ,  $N = 21$ ); Controls ( $r = 0.05$ ,  $p = 0.41$ ,  $N = 23$ ) – or for all participants combined ( $r = 0.09$ ,  $p = 0.24$ ,  $N = 63$ ).

As such, the null hypothesis cannot be rejected.

#### **Hypothesis 5**

**Children with ADHD in the unmedicated condition will give lower estimations of both predicted- and post- performance ratings in comparison with their ratings in the medicated condition and with the ratings of control participants.**

### Predicted Performance Ratings:

Using paired-samples t-tests, a significant difference was found between the two experimental group conditions on the predicted performance rating completed before the task ( $t = 2.15$ ,  $p = 0.02$ ,  $df = 20$ ). Participants with ADHD were significantly more likely to predict a poorer performance in the unmedicated condition in comparison to the medicated condition. The means and standard deviations are presented in table 5.

An independent-samples t-test detected no significant difference between the predicted performance ratings of control participants and participants with ADHD in the unmedicated condition ( $t = 1.57$ ,  $p = 0.06$ ,  $df = 44$ ). However, the predicted performance ratings provided by the ADHD participants during the medicated condition were significantly higher than those given by children in the control group ( $t = -1.86$ ,  $p = 0.04$ ,  $df = 44$ ). See table 5.

### Post-Performance Ratings:

A paired-samples t-test detected no significant difference between the post-performance ratings of children with ADHD across the medicated and unmedicated conditions ( $t = -0.80$ ,  $p = 0.22$ ,  $df = 20$ ).



Similarly, independent samples t-tests detected no significant differences between the control participants and participants with ADHD in the medicated condition ( $t = 0.46$ ,  $p = 0.32$ ,  $df = 44$ ) or between control participants and participants with ADHD in the unmedicated condition ( $t = 0.40$ ,  $p = 0.35$ ,  $df = 44$ ).

A comparison of the mean scores for predicted and post-performance ratings across all three conditions is displayed in table 5 below.

	ADHD-Medicated X (SD) (n)	ADHD-Unmedicated X (SD) (n)	Controls X (SD) (n)
Predicted Performance Rating	2.26 (0.18) (21)	2.00 (0.56) (21)	2.18 (0.14) (25)
Post Performance Rating	2.16 (0.25) (21)	2.20 (0.16) (21)	2.18 (0.15) (25)

Table 5. A Comparison of the Mean Predicted- and Post- Performance Ratings for all Conditions  
(Based on the Transformed Data)

Hypothesis 5 is therefore upheld in part. Children with ADHD in the unmedicated condition did give significantly lower estimations of predicted performance ratings in comparison with their ratings in the medicated condition. However, as there was no significant difference between the predicted performance ratings of children with ADHD in the unmedicated condition and those of control children, nor any

significant differences between the three groups on post-performance ratings, the null hypothesis in its entirety cannot be rejected.

## **Hypothesis 6**

**There will be a difference between the participant groups in the self-evaluation ratings of performance:**

- a) In comparison to control participants, children with ADHD will be more likely to attribute their performance to external factors.**
- b) Children with ADHD will be more likely to attribute their performance to external factors in the unmedicated condition in comparison to the medicated condition.**

a) Independent samples t-tests were used to look for any differences between the ratings given by the experimental participants and control groups. Irrespective of experimental condition, no significant differences were detected for any of the self-evaluation ratings of participants – i.e. ease of paying attention, trying hard, task difficulty or luck. (See appendix 7.16 for details of the non-significant results).

b) Paired-Samples t-tests were used to compare the experimental group's performance ratings on the attribution questionnaire across the medicated and

unmedicated conditions. There were no significant differences between groups for any of the self-evaluation ratings. (See appendix 7.16 for details of the non-significant results).

The participant responses to the various questions on the Attribution Questionnaire are shown in figure VI.

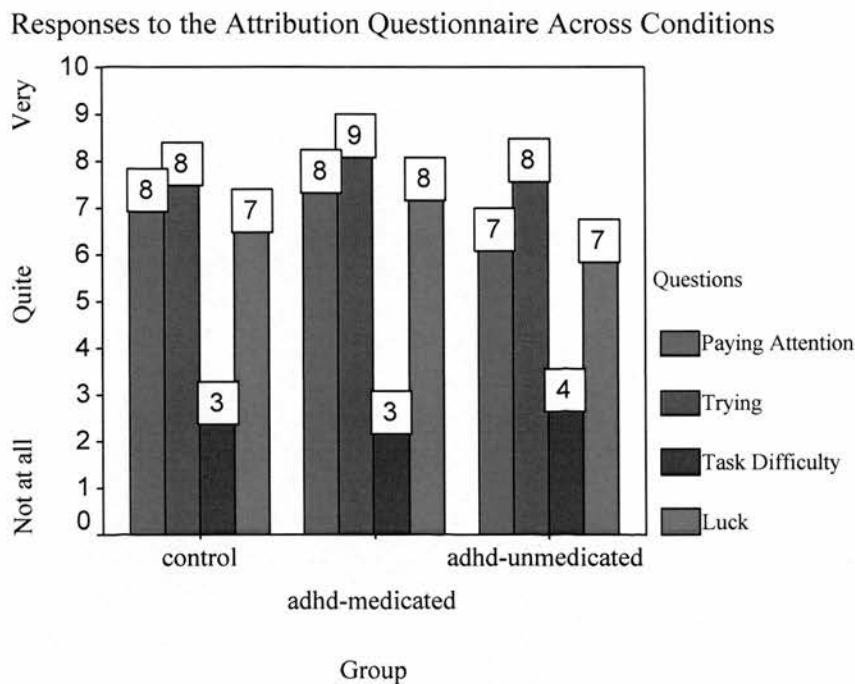


Figure VI. Experimental and Control Participants’ Responses to the Attribution Questionnaire.

Further analysis was performed on the responses provided by participants in the experimental group when ranking the importance of the various factors which contributed to their performance on the Animal Stroop Task. Paired-samples t-tests showed that there were no significant differences between the rankings given for any

of the five variables across the two experimental group conditions (see appendix 7.17). On examining the percentage of responses allocated to each variable, there was a similar breakdown across the two groups, as can be seen from figures VII & VIII.

Ranking Importance of Factors Contributing to Task Performance

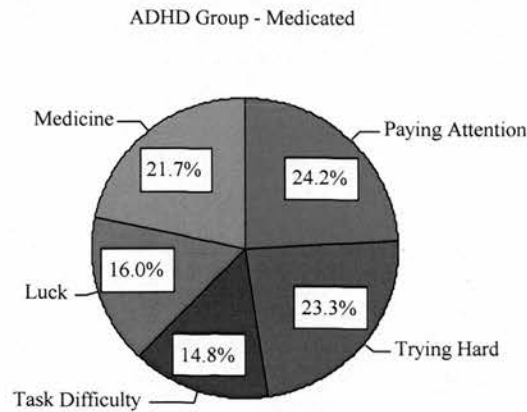


Figure VII. The Importance Ranking of Factors Contributing to the Task Performance of ADHD Participants' in the Medicated Condition

Ranking Importance of Factors Contributing to Task Performance

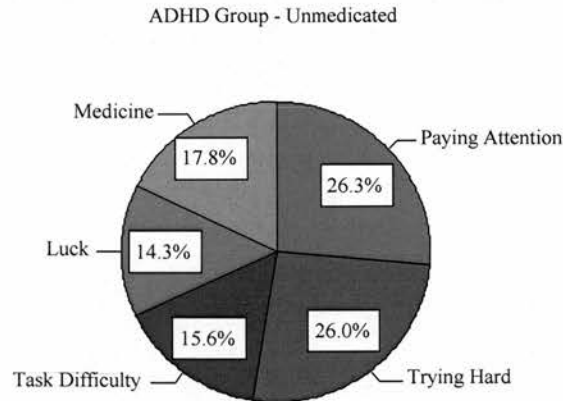


Figure VIII. The Importance Ranking of Factors Contributing to the Task Performance of ADHD Participants' in the Unmedicated Condition

With regard to both parts a) and b) of hypothesis 6, the null hypotheses cannot be rejected.

### 3.3.1 Summary of Hypotheses Testing

**Hypothesis 1** – There was no significant difference between the inhibitory control of children with ADHD and those of children in the control group.

**Hypothesis 2** – In comparison to the unmedicated condition, children with ADHD showed a significant improvement in inhibitory control following their prescribed methylphenidate medication.

**Hypothesis 3** - There was no significant difference between the locus of control scores of children with ADHD and children in the control group

**Hypothesis 4** – There was no significant relationship between locus of control and inhibitory control in any of the participant groups.

**Hypothesis 5** – The predicted performance ratings of children with ADHD were significantly lower in the unmedicated condition compared to their ratings in the medicated condition. However, there was no significant difference between the predicted performance ratings of children with ADHD in the unmedicated condition and those of control children, nor any significant differences between the three groups on post-performance ratings.

**Hypothesis 6**            **a)** Children with ADHD and children in the control group did not differ significantly in the degree to which they attributed their performance to external factors.

**b)** There were no significant differences between the degree to which children with ADHD attributed their performance to external factors across the medicated and unmedicated conditions.

All non-significant results are displayed in appendices 7.16 – 7.17.

## 4. Discussion

This study examined whether methylphenidate is effective in improving response inhibition in children with ADHD, using a newly developed measure of response inhibition – the Animal Stroop Task (Wright et al., 2003). In addition, the study looked at children's own attributions for their level of response inhibition across a variety of variables, including medication status.

The discussion will initially outline the results in relation to each of the hypotheses and discuss these in turn. This will be followed by a consideration of the methodological limitations as well as clinical and ethical implications of the study, before ending with suggestions for further research.

### 4.1. Interpretation of the results

**Hypothesis 1** – There was no significant difference between the inhibitory control of children with ADHD and those of children in the control group. This suggests that children with ADHD (irrespective of whether they are being treated with methylphenidate medication) do not differ from children without ADHD with regard to their level of inhibitory control. As such, the null hypothesis could not be rejected.

**Hypothesis 2** – In comparison to the unmedicated condition, children with ADHD showed a significant improvement in inhibitory control following their prescribed methylphenidate medication. This suggests that methylphenidate is effective in improving the inhibitory control of children with ADHD and, as such, hypothesis 2 was upheld.

**Hypothesis 3** - There was no significant difference between the locus of control scores of children with ADHD and children in the control group. This suggests that children with ADHD are no more likely to attribute their experiences to external factors than normally developing children are. The null hypothesis could, therefore, not be rejected.

**Hypothesis 4** – There was no significant relationship between locus of control and inhibitory control in any of the participant groups. This suggests that children with a more internal-based locus of control are no more able to manage their inhibition than children with a more external locus of control. The null hypothesis could, therefore, not be rejected.

**Hypothesis 5** – The predicted performance ratings of children with ADHD were significantly lower in the unmedicated condition compared to their ratings in the medicated condition. Furthermore, children with ADHD in the medicated condition gave significantly higher predicted performance ratings than children in the control group. In contrast, there was no significant difference between the predicted performance ratings of children with ADHD in the unmedicated condition and those of control children, nor any significant differences between the three groups on post-performance ratings. This suggests that, when medicated, children with ADHD tend

to predict a better performance compared to situations where they have not received medication, and in comparison to normally developing children. With regards to actual-performance, however, all groups rate their performance comparably. Thus, despite hypothesis 5 being upheld in part, the null hypothesis, in its entirety, could not be rejected.

**Hypothesis 6**      **a)** Children with ADHD and children in the control group did not differ significantly in the degree to which they attributed their performance to external factors.

**b)** There were no significant differences between the degree to which children with ADHD attributed their performance to external factors across the medicated and unmedicated conditions.

This suggests that, irrespective of medication status, children with ADHD are no more likely to attribute their task performance to external factors than normally developing children. The null hypothesis could, therefore, not be rejected.

## **4.2 Discussion of the Results**

The findings of the various hypotheses will be discussed in relation to each variable examined and their reference to previously published research considered throughout.



#### **4.2.1 Response Inhibition**

##### **Hypothesis 1**

Hypothesis 1 stated that, in comparison with control participants, children with ADHD would show more impaired response inhibition, as measured by their 'reaction time difference' scores, on the Animal Stroop Task (Wright et al., 2003). However, statistical analysis showed that there were no significant differences in Reaction Time Difference Scores between either the ADHD-medicated and control groups or between the ADHD-unmedicated and control groups, suggesting that children with ADHD (irrespective of whether they are being treated with methylphenidate medication) do not differ from children without ADHD with regard to their level of inhibitory control.

This goes against the now wide-spread theory of response inhibition as the central deficit of ADHD (Barkley, 1997) and is in opposition to previous research which contends that, in comparison to controls, children with ADHD have significant impairments in inhibitory responses across go/no-go tasks (Booth et al., 2005; Hartung et al., 2002; Charman et al., 2001; Iaboni et al., 1995; Shue & Douglas, 1992), the continuous performance task (Halperin et al., 1990), the stop signal task (Schachar & Logan, 1990) and Stroop interference tasks (Gorenstein et al., 1989; Grodinsky & Diamond, 1992; Houghton et al., 1999).

There could be a number of possible explanations for why the current study failed to find a difference between the inhibitory control of children in the control and experimental groups. The majority of the studies that document deficits in response inhibition in children with ADHD use 'Go/NoGo' (e.g. Shue & Douglas, 1992; Booth et al. 2005; Rubia et al. 2001) and 'Stop' paradigms (Oosterlaan et al., 1998). However, as mentioned previously, there are a number of limitations with these measures. Such tasks comprise pure measures of motor inhibition and simple motor reaction times may not be so well-learned to the extent that they necessitate demands on inhibition in order to prevent the response (Wright et al., 2003). It could be that while these tasks provide a measure of *motor* inhibition, they may fail to tap into inhibitory control more broadly e.g. across a cognitive and behavioural level (Wright et al., 2003). On the other hand, measures such as the Stroop task rely on vocal responses. Such reactions may be better established and, consequently, more automatic, making higher demands on inhibitory control in general.

Indeed, studies measuring inhibitory control with the colour-word version of the Stroop task often fail to find impairments in response inhibition in children with ADHD (e.g. Kerns et al., 2001; van Mourik et al., 2005). Such results could be interpreted as evidence that, once the motor component of response inhibition is removed, children with ADHD do not display any deficit in inhibitory control. Nonetheless, other researchers have demonstrated such deficits by using the same Stroop task (e.g. Houghton et al., 1999; Pennington & Ozonoff, 1996; Grodinsky & Diamond, 1992). Given the high rates of co-morbidity between ADHD and reading

disorder (August & Garfinkel, 1990), the fact that many of these studies fail to control for reading ability has been put forward as a possible explanation for these conflicting results (vanMourik et al., 2005). However, measures such as the Animal Stroop task, which use pictorial images rather than words, should be devoid of such shortcomings.

As such, the failure of the current study to find an inhibitory control deficit in children with ADHD may be more representative of Kerns et al.'s (2001) contentions that different tasks may tap different components of inhibition and that not all levels of inhibition are necessarily impaired in children with ADHD (Kerns et al., 2001). Thus it is possible that inhibitory control, as measured by the Animal Stroop, might not be affected by ADHD. It may, then, have been useful for this study to have employed more than one measure of inhibitory control in order to assess the potential for different components of inhibition more thoroughly.

Alternatively, these results may offer further support for the 'delay aversion' hypothesis put forward by Sonuga-Barke and colleagues (1996). The delay aversion hypothesis argues that the cognitive deficits thought to be shown by children with ADHD could actually be more motivational in nature – i.e. children with ADHD are averse to delay. As mentioned previously, Sonuga-Barke et al. (1994) highlighted that studies measuring inhibitory control are often effected by trial constraints (i.e. as soon as one trial ended another began) and, therefore, are confounded with delay. When these experiments have been repeated in such a manner that early or impulsive

responses have no influence on delay, the responses from children with ADHD are comparable with those of controls (Sonuga-Barke et al., 1996). The trials in the Animal Stroop task are of equal length, irrespective of participant's speed of responding. Thus, the current lack of discrepancy between the inhibitory control of children with ADHD and children in the control group may be due to the fact that, as participants could not opt to minimise the delay by acting more impulsively, there was little motivational incentive for children with ADHD to respond more rapidly.

Nonetheless, these explanations fail to account for the initial findings of Wright et al. (2003) who, with reference to the behavioural data of a large sample of children aged between 3 and 16 years, suggested that the Animal Stroop appeared to identify those at risk of hyperactive symptomatology (Wright et al., 2003). It should be noted, however, that Wright et al's (2003) findings were based on data gathered from a *school-based* sample and only utilised behavioural data gathered from teachers rather than the cross-situational information required in considering a diagnosis of ADHD. Thus, the contrast in findings with those of the current study may suggest that using the Animal Stroop task as a measure of inhibitory control is not sufficient in differentiating between children with a *clinical diagnosis* of ADHD and normally developing children. Given that a deficit in response inhibition is considered to be a cardinal feature of ADHD (Barkley, 1997), however, it seems peculiar that the Animal Stroop Task was less, rather than more, discriminating in children with ADHD.

## **Hypothesis 2**

Given the above failure to detect any difference in the inhibitory control of children with and without a diagnosis of ADHD, it may be surprising that analysis of hypothesis two revealed that, in comparison to the unmedicated condition, children with ADHD showed significant improvements in inhibitory control after receiving their methylphenidate medication. These findings fit with the limited, previous reports of methylphenidate ameliorating deficits in response inhibition as measured by a Go/No-go task (Broyd et al., 2005) and by the colour-word version of the Stroop task (Everett et al., 1991). Such improvements in inhibitory control would also tie in with the more general reports of stimulant medication improving both the classroom and social behaviour of children with ADHD (Miller et al., 1998; Pelham et al., 1998), as well as their performance on attentional measures (Hood et al., 2005) and various other executive functioning tasks (Kempton et al., 1999).

However, the current findings go against the results of a more recent study by Scheres et al. (2003) who examined the effects of methylphenidate in relation to the different components of response inhibition. While methylphenidate produced significant improvements in the inhibition of a prepotent response and partial improvements in the inhibition of an on-going response, there was no significant effect of methylphenidate on interference control (as measured by both the Stroop Colour-Word Test and the Eriksen Flanker Task).

It may be possible that the discrepancy in results reflects the different types of Stroop measures employed. Scheres et al. (2003) employed the colour-word version of the Stroop. Due to the automatic reading skills required in this task, only children aged 8 and above were tested. However, reading ability was not controlled for directly and, given the aforementioned increased rates of co-morbidity between ADHD and reading disorder (August & Garfinkel, 1990), it is possible that reading deficits within this sample may have rendered the results of the colour-word stroop inaccurate.

Alternatively, the contrast in results could be due to the differing levels of methylphenidate received by the children in each study. Scheres et al. (2003) examined the effects of methylphenidate dose specifically by employing four different treatment conditions – placebo, 5mg of methylphenidate, 10mg of methylphenidate and 15/20mg of methylphenidate. Although Scheres et al. (2003) found no effects of medication dosage on any of the response inhibition measures, many of the children with ADHD in the current study were receiving doses of methylphenidate-based medication that exceeded a 20mg dose. Indeed, other studies have reported an optimal response to medium or high doses of methylphenidate in cognitive tasks (e.g., Douglas et al., 1988), yet Tannock et al. (1995) found that inhibitory performance declined in their high dose condition compared to the medium dose condition. In contrast, a more recent review examining the effects of low and high doses of methylphenidate on cognitive tasks reported there was no evidence to support an optimal lower dose (Rapport & Kelly, 1991). Due to the

assortment of different methylphenidate-based prescriptions received by the children with ADHD (e.g. long- vs. short-acting preparations), along with the various timings at which these were taken, it was not felt possible to accurately control for the effects of methylphenidate dosage in the current study. Thus, it is not possible to ascertain whether the dosage level of methylphenidate had an effect on the inhibitory control of participants with ADHD.

It may also be worth noting that all participants within the experimental group of the current study had been receiving medication for a minimum period of 4 months. This is in contrast to some of the previous research in which participants are naïve to medication and subsequently experience a period of titration before the study commences. Although these titration periods are relatively extensive (e.g. four weeks in the case of Scheres et al., 2003), it is possible that the length of time participants have been receiving medication has an effect on any subsequently experienced improvements in response inhibition. Indeed, while Scheres et al. (2003) found no improvement in response inhibition following methylphenidate administration, others have reported significant improvements in the inhibitory control (as measured by the Stroop Colour-Word Test) of hyperactive children following a year of treatment with methylphenidate (Everett et al., 1991). Again, due to the variety in the length of time that participants had been receiving psychostimulant medication (i.e. between 4 and 84 months), changes in medication - dose and type - that had occurred within this time, alongside the different doses and

types of medication prescribed presently, it was not felt possible to accurately control for the length of time on medication in the current study.

Given the contrast between results, however, it may be possible that any enhancing effects of methylphenidate on the interference control component of response inhibition are subject to influence by the dose and type of medication, in addition to the length of time medication has been received.

While Everett et al. (1991) did report significant improvements in the inhibitory control of hyperactive children subsequent to methylphenidate treatment, these children still performed more poorly than normal controls. In contrast, despite failing to reach statistical significance, after receiving methylphenidate, children with ADHD in the current study actually demonstrated superior inhibitory control than participants in the control group. The pattern of results detected was that medicated children with ADHD displayed the best inhibitory control, followed by children in the control group, with unmedicated children with ADHD demonstrating the poorest performance (see figure IV and table 4 of the results section).

This is an interesting finding, suggesting that the effects of methylphenidate not only improve inhibitory control in children with ADHD but actually elevate such skill to a level which is above average. This, perhaps, raises an ethical concern in that medication may be being used to alter behaviour in children with ADHD beyond that which is typical of normally developing children.



Given the highly significant improvement in the inhibitory control of children with ADHD between the unmedicated and medicated condition, along with reports that stimulants appear to have similar behavioural effects in normal children (Rapoport & Inoff-Germain, 2002), it would have been interesting to examine the effects of medication on response inhibition in children within the control group. Such investigation would have allowed for a comparison of any level of improvement in inhibitory control between the two groups. However, given the ethical concerns surrounding this notion, along with the constraints of the current research, it was not felt possible to pursue this issue further.

#### **4.2.2 Locus of Control**

##### **Hypothesis 3**

Statistical analysis of hypothesis three showed that there was no significant difference between the locus of control scores of children with ADHD and children in the control group. This contradicts previous research reporting that children with ADHD have significantly higher external locus of control (Lufi & Parish-Plass, 1995; Linn & Hodge, 1982) and are consequently more likely to attribute their experiences to external, uncontrollable factors (Hoza et al., 1993).

In the current study, children with ADHD completed the locus of control scale in the medicated condition only. Given the failure to replicate the previous findings of

increased externality in the locus of control of children with ADHD, it could be surmised that medication has a direct influence on a child's view of their level of control, causing them to feel more in control of their own behaviour and consequently perceiving external factors as being less influential in the experiences they have. However, although participants in Lufi & Parish Plass' (1995) study were not (currently or previously) receiving stimulant medication, Linn & Hodge (1982) reported similarly elevated external scores in their sample of hyperactive males, the majority of which were receiving drug treatment. Moreover, given previously published claims that locus of control is a stable personality trait (Reich et al., 1997), and reports that children with ADHD are more externally orientated regardless of medication status (Lufi & Parish-Plass, 1995), it was not felt necessary for participants to complete the locus of control scales in both the medicated and unmedicated conditions. Nonetheless, in view of the failure to replicate findings of increased external locus of control in children with ADHD, it may have been useful to examine whether the locus of control scores of the experimental participants were indeed influenced by the medication condition of the current study.

It should also be noted that although the same measure of locus of control was utilised across all three studies, the participants in Linn & Hodge's (1982) study and Lufi & Parish-Plass' (1995) study were, respectively, comprised of 16 boys from New Mexico and 28 Israeli boys. While the Locus of Control Scale for Children is considered suitable for use across a wide variety of countries (Nowicki & Strickland, 1973), in view of the fact that all participants in the current study were of British

origin, it may be possible that cultural differences were influencing the responses provided.

#### **Hypothesis 4**

Given people with a more internally-based locus of control are more likely to perceive a connection between their behaviour and the outcome (Nowicki & Kalechstein, 1997), it was hypothesised that children who have a more internally based locus of control may demonstrate better inhibitory control (Hypothesis 4). However, correlation analysis showed that there was no significant relationship between locus of control and inhibitory control in any of the participant groups. This is perhaps unsurprising given that children with ADHD were comparable to children in the control group with respect to both inhibitory control and to locus of control. However, such findings could also be interpreted as evidence that inhibitory control is not subject to modification by any degree of conscious personal control and, as such, may constitute a more biologically-based ability. Tentative support for such contentions may also be offered by the results of a recent study reporting that, in children with ADHD, perceived control and behavioural difficulties are not strongly related (Costigan, 2000).

### 4.2.3 Behavioural Attributions

#### **Hypothesis 5**

Analysis of hypothesis five revealed that the predicted performance ratings of children with ADHD were significantly lower in the unmedicated condition compared to their ratings in the medicated condition. While this may be interpreted as evidence that children with ADHD are significantly more likely to under-predict their level of performance during times they are not receiving medication, it is worth noting that there was no significant difference between the predicted performance ratings of children with ADHD in the unmedicated condition and those of control children. This would suggest that children with ADHD, without medication, actually predict their performance at a level comparable to that predicted by normally developing children. Conversely, the predicted performance ratings provided by the participants with ADHD during the medicated condition were significantly higher than those given by children in the control group, suggesting that, prior to undertaking tasks when medicated, children with ADHD have more confidence in their ability to perform than normally developing children do. Overall then, although children with ADHD predict poorer performance levels during periods where they are not receiving medication, these predictions are still comparable with that of normally developing children. On the other hand, the performance predictions of children with ADHD who are receiving medication are significantly higher than normally developing children and, as such, may be unrealistic.

Nonetheless, on examination of the original data, however (see appendix 7.18 for original data and table 5 of the results section for transformed data), it becomes apparent that, while the ratings of children with ADHD in the unmedicated condition and the ratings of children in the control group tend to increase following task completion, there is very little difference between the pre- and post- performance ratings of children with ADHD in the medicated condition. It could be then, that both controls and children with ADHD without medication, are more likely to give lower predicted ratings of their performance as a cautionary measure, yet, after completing the task, feel sufficiently confident to increase this rating. On the other hand, children with ADHD who are receiving medication may feel more confident in their ability to accurately predict their performance from the outset and, as such, feel less need to under-predict their performance initially.

These findings fit, to some degree, with a growing body of research contending that children with ADHD may be characterised by overly optimistic self-perceptions (Hoza et al., 2000). For instance, when reporting self-perceptions at the onset of a treatment programme, the ratings provided by boys with ADHD, in terms of academic, social and athletic functioning, were statistically 'indistinguishable' from those of control participants (Hoza et al., 1993). Expanding on this, Hoza et al. (2000) found that boys with ADHD rated their own performance on a social task significantly more favourably than control boys did. Indeed, this 'positive illusory bias' was sometimes even more extreme following a negative social experience.

From this finding, the authors suggest that inflated self-perceptions may be one way in which boys with ADHD try to cope with failure. Similar contentions have also been put forward by Diener and Milich (1997) who propose that enhanced self-perceptions serve a protective function for children with ADHD by counteracting feelings of inadequacy.

Indeed, even amongst non-disordered individuals, a positive illusory pattern is normative and it has been suggested that,

*“positive illusions....may be especially apparent and adaptive under circumstances of adversity, that is, circumstances that might be expected to produce depression or lack of motivation”* (Taylor & Brown, 1988, p. 201).

It may be possible, then, that for children with ADHD, inflating their predictions of performance provides them with a protective, buffering measure against the possibility of potential failure. Nonetheless, this explanation fails to account for why these children's predictions become comparable with those of controls when they are no longer receiving medication. Nor does it account for why there are no significant differences between the post-performance ratings of children with ADHD, either across medication conditions or in comparison with controls.

Milich (1994) proposed that medication may somehow ameliorate potential failure experiences, while facilitating a healthier, more adaptive style of responding. Indeed, having the ability to believe that something will go well forms the basis of the widely-advocated use of ‘positive self-talk’ as a coping strategy throughout the

child and adolescent literature (e.g. Webster-Stratton, 2006; Carr, 1999). It is possible then, that when faced with a potential failure experience, medication afforded participants with ADHD the capacity to generate enhanced self-perceptions, an ability that was not afforded in the unmedicated condition. Nonetheless, after completing the task and viewing it as a successful experience (all participants reported they had done a good job), there was no longer the same need for this buffer, hence the post-performance ratings of children with ADHD who were receiving medication remained at a level which became comparable to their post-performance ratings in the unmedicated condition and with the ratings of controls.

Although a plausible explanation, it should be noted that the post-performance ratings of all participants, as well as the pre-performance ratings of the ADHD-medicated group, tended to be fairly high on the 10-point likert scale. Thus, these results may be subject to ceiling effects, which could be masking the potential for ratings to have risen further.

### **Hypothesis 6**

Extending this examination of self-evaluations, analysis of hypothesis six revealed that children with ADHD and children in the control group did not differ significantly in the degree to which they attributed their performance to external factors. As such, participant's perceptions of (a) how easy they thought it had been to pay attention; (b) how hard they felt they had tried; (c) how lucky they felt they

had been; and (d) how hard they thought the task was, were comparable across the control and experimental groups. Furthermore, these perceptions remained comparable, regardless of whether or not children with ADHD were in the medicated or unmedicated condition. In addition, irrespective of medication condition, there was no difference in the degree of importance participants with ADHD attached to each of the five variables (attention, trying, task difficulty, luck and medication) with respect to the overall influence they had on performance.

These results fit with the study's earlier finding of comparable locus of control scores between the experimental and control participants, thus, together, could be interpreted as evidence that, contrary to popular belief (e.g. Hoza et al., 1993; Lufi & Parish-Plass, 1995), children with ADHD are not more likely to attribute their performance to external factors or to view their experiences as being outwith their personal control. Indeed, the results of the current study directly replicate some of the findings of an earlier study by Milich et al. (1989), in which, despite yielding significantly superior scores on the Continuous Performance Task on medication compared to placebo, there were no significant differences in the mean self-evaluations and attribution responses of participants with ADHD across the two conditions. Furthermore, in the current study, no evidence was found to support contentions that medication may produce predominantly external or medicated-related explanations for performance (e.g. Rosen et al., 1985). In fact, in comparison to the other two, more internal factors considered, participants were less likely to choose 'medication', 'luck' or 'task difficulty' as being important to their overall



performance. This pattern was consistent regardless of medication status (see figures VII & VIII of the Results section).

As mentioned earlier, other researchers have found a difference in the attributions made by children with ADHD in comparison to those of control children. For instance, Hoza et al. (2000) reported that boys with ADHD were more likely to attribute task success to external, uncontrollable factors such as task ease and luck, while children in the control group were more likely to attribute failure to internal factors such as not trying hard enough. However, this study assessed participants' attributions about a social task in which they were required to 'get-acquainted' with another child. It may be possible that the process of making attributions for our behaviour varies as a function of whether the task makes social or academic demands. Given that the task employed in the current study was largely academic-based, the difference in the nature of the two tasks may provide some explanation for the discrepancy in results.

Milich et al. (1989), however, went onto report a finding of increased correspondence between the actual performance and self-evaluations of participants in the medicated condition, compared to the placebo condition. They interpreted this as evidence that medication may enhance participants' ability/motivation to monitor their own behaviour, consequently leading them to develop better task strategies (Milich et al., 1989). Unfortunately, due to measurement differences in the way that

actual and self-perceived performance was scored in the current study, it was not possible to compare the two variables directly. This is a considerable methodological weakness as such comparisons would have allowed a determination of the degree of accuracy with which all participant groups were rating their performance and may have offered further potential for explaining the findings reported.

Nonetheless, the current results suggest that perceptions of performance in children with ADHD do not differ from those of control children or across medication status. As such, these results contradict contentions that medication may lead children with ADHD to attribute their behaviour to external factors (e.g. the drug) while viewing their own efforts/abilities as having a relatively minor role (Bugental et al., 1977).

#### **4.2.4 Summary**

The study found that, irrespective of medication status, children with and without ADHD did not differ in respect of the following: their level of inhibitory control, their locus of control scores, their self-evaluated performance or the attributions made for their performance. Furthermore, no relationship between locus of control and inhibitory control was detected. Nonetheless, methylphenidate did significantly improve the inhibitory control of children with ADHD. It is suggested that these findings may be the result of a number potential factors including motivational aspects, the possibility that different tests tap different components of response

inhibition (Kerns et al., 2001), as well as the potential impact of a positive illusory bias in children with ADHD. Variations in the types of measures employed, dose and type of medication received, and cultural differences between study participants are offered as potential explanations for discrepancies between the current findings and those of previous research. The next section will examine the methodological limitations of the study and the extent to which these may have influenced the results.

### **4.3 Methodological Considerations**

#### **4.3.1 Limitations of the Measures**

##### **Estimated Full Scale IQ**

Full-scale IQ was estimated using a shortened version of the Wechsler Intelligence Scale for Children, UK (WISC-III UK, 1991). This was comprised of one verbal subtest (Vocabulary) and one performance subtest (Block Design), with a Full-Scale IQ score being estimated using the method described by Sattler (1992). Although the validity of this short form is high ( $r = 0.906$ ), a number of methodological problems question the utility of short-forms (Sattler, 1992). Even those with high validity have been found to misclassify people and limit the opportunity for profile analysis (Sattler, 1992). As the current study found no significant group differences in full-scale IQ estimates, this variable was excluded from analysis. However, it should be noted that, had the research been subject to fewer constraints, it may have been useful to assess children's IQ more accurately using the full-version of the scale.

## **The Animal Stroop Task**

As Schachar and Logan (1990) expressed,

*“...no widely accepted measure of inhibition exists”* (Schachar & Logan, 1990, pg. 711).

The Animal Stroop was selected on the basis of its recognition as a reliable and valid measure of inhibitory control (Wright et al., 2003) and, being pictorial-based, it had the particular advantage over other Stroop-type measures in that it did not require a certain level of reading ability in order to yield reliable scores. However, there may be potential limitations of the Animal Stroop Task with regards to its use in the current study.

Wright et al. (2003) advocate that the Animal Stroop Task provides a robust measure of inhibitory function across the age range of three to sixteen years. However, they do warn that, while the content domain of the task (i.e. animal picture naming) is sufficiently easy for younger children, it may be a less sensitive measure of impulse control in older children (Kail, 1991). The authors report that a Stroop-like effect did remain, even in their 16-year old participants, but do caution that there may be floor and ceiling limitations on its validity. The participant sample of the current study comprised children up to the age of 14 years and with this, alongside the researcher's own observations of increased task ability in older children, it is possible that ceiling effects amongst the older participants may be masking a difference between the inhibitory control of participants with and without ADHD.

Although the measure only took approximately ten minutes to complete, many of the children, especially those with ADHD, became distracted during the task. As such, their response times were often quite variable and, in some cases, the number of omission and response errors was fairly high. This resulted in a smaller number of valid trials being available for the calculation of inhibitory control, thus reducing the reliability of these measures for some participants. Nonetheless, given attentional difficulties are one of the main behavioural features of ADHD, it would have been useful to examine this more thoroughly, perhaps by looking at whether there were differences between the number of omission and commission errors accrued in the different participant groups and experimental conditions. However, due to a lack of published material regarding the Animal Stroop Task, it was not clear what these error rates were specifically measuring. As such, it was considered best to exclude them from further analysis.

### **Locus of Control**

As mentioned, locus of control was measured using the Locus of Control Scale for Children (LCSC) (Nowicki & Strickland, 1973) for participants aged nine and over, while the Preschool and Primary Nowicki-Strickland Internal-External Scale (PPNSIE) (Nowicki & Marshall, 1974) was used for those below nine years of age. Used together, these scales allowed for a parallel, age-appropriate, reliable and valid measure of locus of control for children across the age-range of the sample (Nowicki

& Strickland, 1973; Nowicki & Marshall, 1974). Indeed, the Nowicki-Strickland scales are considered to be

*“...one of the most widely used [scales]”* (Furnham & Steele, 1993, pg. 462).

However, rather than classifying people as having an ‘external’ or ‘internal’ locus of control, the scale measures the degree of internality and externality on a continuum. As such, the nature of the data dictated that only correlational analyses could be carried out rather than tests of difference.

In addition, some of the recent research has moved away from the internal-external dichotomy, claiming that a more differentiated view is needed in order to fully investigate the developmental aspects of control perceptions (Skinner & Connell, 1986). As such, a number of multi-dimensional scales have now been developed. For instance, Connell (1985) developed the ‘Multidimensional Measure of Children’s Perceptions of Control’ in which three dimensions of control are independently assessed; ‘internal’, ‘powerful others’, and ‘unknown’. Each of these sources of control is further assessed within three behavioural domains: ‘cognitive’, ‘social’, and ‘physical’. Connell (1985) claims that multidimensional measures provide a richer ‘idiographic’ representation of children’s perceptions of control. It is possible that by using a multi-dimensional scale, there may have been more opportunity to assess children’s perceptions of control in more depth. However, given the constraints of the current research, alongside the fact that the majority of these scales are not suitable for use with younger children, the Nowicki-Strickland Scales appeared to be the most appropriate tools to employ as a measure of Locus of Control.

### **Self Evaluation and Attribution Questionnaire**

Due to a lack of standardised measures relating to the specifics of the tasks employed, the researcher developed a questionnaire to assess participants' evaluation and attributions for their performance on the Animal Stroop task. While this assessment was based on previous research examining attributions (Milich et al., 1989), the reliability and validity of the measure remains unknown.

In retrospect, a number of features of the questionnaire could have been improved. While the majority of participants were considered to fully understand all elements of the questionnaire, some of the younger children appeared to have difficulty with the concept of rating using a Likert scale. The researcher tried to limit this effect by using a pictorial representation of the scale – i.e. an illustration of a thermometer where the temperature gauge increased, by intervals of one, from 0 – 10 (see appendix 7.19) – and by explaining the process of rating to the child using developmentally appropriate language. Despite each child confirming that they understood, those participants (particularly the younger children) whose comprehension of what was required seemed less clear, tended to opt for the extreme point of the scale – i.e. number 10. As such, the rating data may have been subject to ceiling effects. However, a number of researchers note that, when asked to use Likert rating scales, younger children seem to respond in an extreme manner (i.e. selecting the options placed at either end of a response continuum) (e.g. Chambers & Craig, 1998; Goodenough et al., 1997). This is supported by Piagetian theory



contending that young children characteristically engage in dichotomous thinking (Gelman & Baillargeon, 1983). As such, this extreme pattern of responding amongst the younger participants was, perhaps, unavoidable.

Some of the questions themselves could also have been altered. For instance, basing it on Milich et al.'s (1989) study, children were asked to rate how easy it had been to pay attention and to rank how important they felt paying attention had been to their overall performance on the task. Enquiring about perceptions of attention was particularly relevant to Milich and colleagues who were assessing a group of boys with ADHD on the Continuous Performance Task – a test of sustained attention. Although attentional difficulties are one of the cardinal features of ADHD, given that the current study aimed to specifically assess inhibitory control, it may have been more valuable to replace the questions pertaining to attentional ability with ones that enquired about participants' perceived response inhibition e.g. 'How easy did you find it to name the animal's body instead of the head?'. Inclusion of this question may have allowed for more direct investigation of participants' perceptions of their own inhibitory control.

Participants in the experimental group were asked directly about any effects they felt their medication may have had on their task performance and some comparison across the medicated and unmedicated conditions was afforded by including 'medication' as a factor in the list of variables that participants were asked to rank in order from least to most important. While the importance attached to 'medication'



remained relatively stable, irrespective of condition, it may have been useful to have included additional questions to allow more detailed comparisons of the perceived effects of medication. For instance, in the unmedicated condition, participants were asked if they felt they would have performed 'better', 'worse' or 'the same' had they taken their medication as usual. Overall, the majority of participants (52.4%) felt they would have performed to an equivalent standard after taking medication. Almost 40% of participants with ADHD believed they would have done better on the Animal Stroop task had they been able to take their medication, while just under 10% reported that they would have done worse. However, participants were asked this question in the unmedicated condition only and, as such, there was no scope for examining whether these perceptions might have been different had participants taken their medication. It may, therefore, have been useful to have included a modified version of this question which could have been posed to participants in the medicated condition – e.g. 'If you had not had your medicine as normal this morning, do you think you would have performed 'better', 'worse' or the 'same?'. Similarly, participants in the medicated condition were asked to rate how much they felt their medication had helped on a scale of 1-10. Including a corresponding question in the unmedicated condition (e.g. 'If you had had your medication as normal this morning, how much do you think it would have helped you with the task?'), would again have allowed more of a comparison of participant views across conditions.

Lastly, rather than asking participants to give a general rating of their overall performance out of 10, it may have been more appropriate to ask them to pre- and post-estimate the number of mistakes they felt they would make/made. This could

have allowed for a more direct comparison of perceived- vs. actual- performance. Nonetheless, given the aforementioned concerns about what the error data actually measures, any such results would have required cautious interpretation.

Despite the flaws highlighted above, the researcher's need to self-design this questionnaire was necessitated by the lack of standardised measures relating to the specifics of the task employed. It should also be noted that while, in some respects, a novel measure, the assessment was essentially based on the questions used in previous research of a similar nature (i.e. Milich et al., 1989). Had the current research not been subject to time constraints (see subsequent discussion), it may have been beneficial to have conducted a pilot study so that any such weaknesses could have been detected and modified accordingly. Overall, however, the questionnaire employed did cover the main areas under investigation and was considered appropriate for use with the large majority of participants.

#### **4.3.2 Limitations of the Sample Size**

The ADHD database contained 98 names of children deemed suitable to take part in the study, therefore all 98 families were contacted and invited to participate. In order to achieve statistical power for the between subjects tests of difference, 20 participants were required, while only 12 participants were required for the within subjects tests of difference (Clark-Carter, 2004) - response rates of 20.41% and 12.24% respectively. With an overall response rate of 25.51%, the required number of participants was recruited. This remained the case even after 4 experimental

participants were excluded due to incomplete data, reducing the total number of experimental participants to 21.

It should be noted, however, that two participants were removed from each of the ADHD group (medicated condition) and the Control group due to outlier scores on the inhibitory control measure of the Animal Stroop. While these scores needed to be removed in order for the groups to be matched on the inhibitory control task, it meant that in analyses examining inhibitory control, the experimental group (medicated condition) was reduced to 19 and, as such, did not meet the 20 participants required for the between subjects tests of difference. In these particular analyses then, the number of participants within the medicated condition of the experimental group was insufficient to reach statistical power.

#### **4.3.3 Time Constraints**

Although the required number of participants was, for the most part, achieved, it may have been possible to recruit more participants had the study not been affected by the considerable time constraints on the data collection phase of this study.

For instance, the process of acquiring ethical approval is one which is lengthy and often time-consuming. Had this not been a factor in delaying data collection, there may have been more time to recruit additional participants and/or look to extending the research into another service area, e.g. Lothian.

A number of other factors may have played a role in the relatively low participant response rate. Given the link between parents with a chaotic parenting style and children with ADHD (Johnston & Mash, 2001), it is possible that families may simply have lost or forgotten to return the forms. While some families were sent the information on more than one occasion, it was not felt practical or ethical to re-send the information to all 98 families. People may also have been put off by the direct testing aspect of the study, with parents and/or children potentially worrying about the level at which they might perform. It is possible that the requirement for participants to be seen twice for relatively lengthy sessions was also a factor that influenced whether people decided to take part. Although attempts were made to make this process as convenient as possible by offering each participant the option of where they would prefer to be seen – i.e. school, the unit or, in some cases, at home - it is possible that the level of commitment these appointments engendered may have deterred people from taking part. Having to skip a dose of medication is a component that may have been influential in the recruitment of participants. While the lack of risk associated with this procedure was detailed in full within the participant information sheet, with both the researcher and the Child and Adolescent Psychiatrist being available for consultation throughout, this factor may have been enough to dissuade people from participating.

Given the response rate amongst the control group was considerably higher (73.53%), it is likely that some of the above factors - specific to participants in the experimental group - were most influential in the process of participant recruitment.

As mentioned previously, had the research not been subject to such time constraints, it may also have been useful to have conducted a pilot study in order to allow for any potential weaknesses detected to be modified accordingly.

#### **4.3.4 Design Limitations**

##### **ADHD Diagnosis**

As mentioned in the introduction, ADHD encompasses three subtypes – ‘inattentive’, ‘hyperactive-impulsive’, and ‘combined’ ADHD (Rappley, 2005). While all participants in the current study had been given a diagnosis of ADHD by a consultant psychiatrist or multi-disciplinary team prior to taking part in the study, they had not been classified as a particular subtype and, as such, the current study did not take into account the potential influence of different subtypes. This, however, may have been an interesting factor to include, allowing for investigation of whether the inhibitory control of participants with ADHD differed depending on their specific subtype of ADHD. It might be expected that those classed as ‘hyperactive-impulsive’ or ‘combined’ types would perform more poorly than those classed as being within the ‘inattentive’ subtype. Nonetheless, classifying participants with ADHD into the different subtypes can be problematic in that it is not necessarily as easy to define children into three clearly distinct subtypes. For instance, children in either the

inattentive or hyperactive-impulsive subtype may be just one symptom below the threshold for the combined subtype, thus specific subtypes may be contaminated by contrasting features of another subtype (vanMourik et al., 2005).

Although children in the experimental group had been given a diagnosis of ADHD, the study did not make use of any independent diagnostic interview or corroborating diagnostic information. It may, therefore, have been beneficial for the study to have included a parent interview, allowing the researcher to gain further information about the child, e.g. by means of administering the Diagnostic Interview Schedule for Children (DISC)-IB (Shaffer et al., 2000) - a structured interview that generates DSM-IV diagnoses. Not only would use of this schedule have allowed the researcher to ascertain which of the ADHD subtypes each participant came under and provided the opportunity for these aspects to be explored further, it would have afforded additional support for the original ADHD diagnosis. Examining the different subtypes of ADHD with regard to both inhibitory control and to the self-perceptions of participants with ADHD may present an area for further research.

Children who were diagnosed with any other significant mental health condition (e.g. autistic spectrum disorder) were excluded from the study, as were children with a learning disability. This was felt to be an appropriate measure to take in order to ensure that participants would be able to sufficiently complete the study's requirements without causing distress. Nonetheless, with an abundance of research

documenting the high incidence of comorbid disorders in ADHD (Moser & Bober, 2002), it is possible that the current sample may not accurately represent the general ADHD population. It should be noted, however, that recent research suggests that comorbid disorders are unlikely to account for the related cognitive difficulties in children with ADHD (e.g. Nigg et al., 1998).

### **Inhibitory Control and Behaviour**

It would have been interesting to compare the results of the inhibitory control measure to behavioural data. Using a school-based sample of participants, Wright et al. (2003) compared participants' behavioural data (collected from teacher-rated Conners ADHD index (Conners, 1996)) to their scores on the inhibitory control measure of the Animal Stroop task. Although correlations suggested an absence of a general relationship between task performance and ratings of behaviour, Wright et al. (2003) went on to categorise the participants into 'high' and 'low' risk groups depending on whether they scored above or below the 75th percentile on Conners ratings of hyperactivity. On the basis of their performance on the Animal Stroop Task, children were also classified as having either 'good' or 'poor' inhibition. These more detailed comparisons suggested that children with high hyperactivity or ADHD ratings performed more poorly on the measure of inhibitory control than those with low behavioural ratings. In addition, children in the poor inhibition group were rated as more hyperactive, more oppositional, and as possessing more symptoms of ADHD than those classed as having good inhibitory control. The authors interpreted these findings as evidence of an association between poor

inhibitory control and a range of behavioural symptoms in a non-clinical sample of hyperactive children.

In view of the promising results of this study, it would have been interesting for the current study to have compared behavioural data with the inhibitory control scores of the experimental group to see whether the relationship between behaviour and inhibitory control was replicated in a clinical sample. However, given the various constraints of the current research, it was not felt possible to include investigation of behavioural data. It may, however, be a valuable area for further research to explore.

### **The ‘Skipping a Dose’ Strategy**

Within the unmedicated condition of the study, children with ADHD were assessed after not having had any methylphenidate for a period of at least 12 hours. Considering the short-half life of methylphenidate, 12 hours should be ample time for any effects of the drug to have died out. However, by using this strategy, all participants were aware that they had skipped a dose and it may be possible that the knowledge of not having had medication was enough to generate changes in the children’s behaviour and consequently their level of inhibitory control. Unfortunately, it was not possible to control for the effects of any such influence within the study. Rather than directly skipping a dose then, it may have been more effective to have administered a placebo drug to all participants prior to testing them in the unmedicated condition. Indeed, other studies have utilised such methods (e.g. Milich et al., 1989). However, using a placebo would not have afforded the assessment of whether there were self-evaluation differences associated with



participants knowing whether or not they were on medication. Furthermore, there are ethical concerns over the use of a placebo and, given the participants with ADHD were all part of a clinical population attending for treatment, using a placebo was not considered appropriate.

### **Motivation**

Poor response inhibition in children with ADHD is often understood as a motivational deficit whereby children with the disorder do not apply the level of effort required to achieve and sustain optimal performance (Oosterlaan et al., 1998). Within this theory, researchers propose that children with ADHD possess an exceptionally strong tendency to seek immediate rewards (Douglas, 1989). It was noted that some of the children in the current study, particularly those with ADHD, did appear to lose interest in the Animal Stroop task and become quite impatient. Both participant motivation and appeal of the task may, therefore, have played a role in the overall results.

The issue of motivation may tie in with the previously mentioned finding that participant response times were often quite variable. Indeed, the majority of children with ADHD had at least one trial that the Animal Stroop task guidance highlighted as comprising ‘unreliable reaction times’ - suggesting that the variability in the individual’s reaction times is greater than that found in the normative sample (Wright, 2006, personal contact). In addition, the number of omission and response errors yielded by some participants was fairly high. While this was also true for some of the control participants, this pattern of responding was more common in

participants with ADHD. Indeed, other studies have reported similar findings of slow, variable and inaccurate responses in children with ADHD (e.g. Kunsti et al., 2001). This pattern fits with a state-regulation theory of hyperactivity (Van der Meere, 1996) which argues that the core problem in hyperactivity relates to a non-optimal state of activation/effort, potentially leading to a lack of consistent effort (Oosterlaan & Sergeant, 1996).

Indeed, some studies investigating inhibitory control aim to increase motivation for effort by informing children when they have responded incorrectly and only analyse the reaction times of trials where responses were correct (e.g. Simpson & Riggs, 2005). Indeed, Douglas & Parry (1983) contend that simple, continuous positive verbal feedback can reduce the variability of reaction times and decrease mean reaction times of children with ADHD. Others go as far as to impose a system of monetary reward and monetary loss (Jennings et al., 1997). As such, providing participants with feedback or some other form of incentive may have encouraged children with ADHD to perform at an optimal level and maintained their motivation to stay focused on the task, as well as reduced the variability of reaction times. In the present study, however, it is possible that a lack of motivation may have resulted in children with ADHD performing more poorly than they might otherwise have done. Given the study was also examining participants' self-perceptions of their performance, it was felt that providing such comment, may influence the responses given on the attribution questionnaire and may, consequently, effect their legitimacy as *participant* perceptions. Further research is needed in order to decipher the degree

to which the activation/effort state of children with ADHD influences their capacity for inhibitory control.

### **The Effect of Behavioural Treatment Programmes**

There is limited research examining the effects of behavioural treatment programmes on the attributions made by children with ADHD. However, it has been shown that self-control plus attributional training for children with ADHD improved their academic and classroom performance, as well as their attributions of personal control (Reid & Borkowski, 1987). Parental attributions for a child's behaviour can also change as a result of behavioural treatment (Johnston & Leung, 2001). Johnston and Leung (2001) found that, with behavioural management, parents viewed the compliance of children with ADHD as being more external and stable, while non-compliance was seen as more controllable but less stable. The current study did not control for whether participants had ever undergone any form of behavioural treatment. As such, it may be possible that other interventions may have had an effect on participants' locus of control and/or the attributions made.

### **4.4 Clinical and Ethical Implications**

A major clinical implication of the current study is that it raises awareness of the need to monitor the effects of medication treatments on response inhibition. Whether medication is improving a child's ability to control their inhibition is an important area as such improvements may aid them in other areas of development, such as attentional ability, social skills etc. Indeed, Tannock et al. (1989) found that, in

children with ADHD, improvements in behaviour and academic performance resulting from treatment with methylphenidate were strongly associated with improvements in inhibitory control, as measured by the stop-signal paradigm. Conversely, monitoring the symptoms of ADHD is also important when we consider the abundance of negative consequences that can develop from the disorder, such as conduct disorder (Taylor et al., 1996). In particular, considering research linking inhibitory control to psychopathology by focusing on the impulsive component of ADHD (Schachar et al., 1993), the monitoring of inhibitory control in relation to applying interventions to help counter such difficulties, may be especially important.

In clinical practice, the effects of medication on children with ADHD are monitored in relation to their effects on observed behaviour. These observations, however, can be fairly subjective. Utilising a variety of measures is important as improvements in response inhibition may not be evident through behavioural observations. Although producing no changes in perceptible behaviour, it is possible that increased inhibitory control may be having more subtle effects that might influence a child's academic ability – a feature more clearly detected by neuropsychological testing. The results of this study highlight the effectiveness of involving neuropsychological tests in monitoring the potential effects of medication on response inhibition. By providing an indication of whether medication would be effective, such tools could also provide useful information to be included in the consideration of whether a child should be given a trial of medication. Thus, being readily available and easy-to-

administer, clinicians should recognise the value of using neuropsychological tests, such as the Animal Stroop, more widely in clinical practice.

The lack of significant difference between the inhibitory control of children with ADHD (irrespective of medication status) and children in the control group was also an important finding, which calls question over Wright et al.'s (2003) original suggestion of using the Animal Stroop task as a potential screening measure to identify children at risk of hyperactive symptomatology.

In addition to the clinical implications of this research, the study also raised some ethical issues for consideration. For instance, the finding that children with ADHD demonstrated superior inhibitory control whilst on medication, not only in comparison to their performance off medication but in comparison to that of participants in the control group<sup>3</sup>, suggests that methylphenidate may actually elevate children's capacity for inhibitory control to an above-average level. As such, the finding highlights ethical concern over the potential for medication to be used as a method of enhancing behaviour in children with ADHD to an extent that is beyond that typical of normally developing children. Given that the behaviour of normally developing children can also be improved by stimulant medication (Rapoport & Inoff-Germain, 2002), this matter could prove to provoke considerable concern.

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<sup>3</sup> It should be noted that the difference between inhibitory control between children with ADHD and children in the control group did not reach *statistical* significance.

Nonetheless, the finding that the self-perceptions and attributions made by children with ADHD did not vary as a function of their medication status, does ameliorate previous concerns that medication may lead children with ADHD to attribute their behaviour to external factors (e.g. the drug) while viewing their own efforts/abilities as having a relatively minor role (Bugental et al., 1977), and diminishes notions of them subsequently becoming reliant on drugs to focus their attention and effort (Rosen et al., 1985).

#### **4.5 Future Research**

Despite the methodological constraints of the current study, in addition to the aforementioned avenues for research already discussed above, the study highlighted a number of other areas which may require further research.

Firstly, the lack of a significant difference between the inhibitory control of participants with and without ADHD was unexpected and one which requires further investigation. Given the current research was the first known study to make use of the Animal Stroop as a measure of inhibitory control with a clinical group of children diagnosed with ADHD, it would be useful to see whether these findings are replicated. In addition, it would be interesting to see how the performance of children with ADHD on the Animal Stroop compares to their performance both on the original, widely-used, Colour-Word Stroop task, as well as to other tests of response inhibition. Such comparisons may aid further exploration of the notion that different tests tap distinct components of response inhibition (Kerns et al., 2001).

Due to the restraints of this research, it was not felt possible to examine any additional relationships between gender and/or age on response inhibition, medication effects and/or attributions for performance. Given the incidence of ADHD is significantly higher in boys (outnumbering the incidence in girls by between 4:1 (Gaub & Carlson, 1997) and 3:1 (Barkley, 1997)), it is not surprising that previous research on ADHD tends to focus on male participants. While studies involving girls with ADHD tend to report that there are no effects of gender on response inhibition (e.g. Daugherty et al., 1993; Kunsti et al., 2001), given the limited amount of research encompassing female participants, it would have been useful to re-examine these contentions within the current sample. Furthermore, exploration of whether gender had any influence over the effects of medication and/or the attributions made by participants may also have been interesting.

Despite inhibitory control in normally-developing individuals being shown to improve throughout childhood (Williams et al., 1999; Wright et al., 2003), this relationship is non-linear - the most rapid improvements in inhibitory control being between the ages of 3 ½ - 5 years (Simpson & Riggs, 2005). There is little research examining the development of this ability in children with ADHD, however, Jennings et al. (1997) reported that, while 10-12 year-old boys with ADHD showed better response inhibition than their 8-10 year-old counterparts, there was no such improvement in a group of control boys. It may well be possible then that, in regards to inhibitory control, children with and without ADHD experience different

developmental trajectories. If children with ADHD are, indeed, slower in their ability to develop such skills, this may help account for some of the other difficulties inherent within the disorder. Further exploration of the relationship between age and response inhibition in children with ADHD would confirm whether this is a possibility.

In addition, Milich et al. (1989) found that children's attributions vary as a function of age – older boys being less likely to attribute success to medication and more to ability. It would, therefore, have been interesting to examine whether the age of participants in the current study had any effect on the attributions they made. Factoring in the potential influence that age may have on the effects of medication, in relation to both inhibitory control and behavioural attributions, is also an area that may warrant further investigation.

While this study looked at participants' own attributions for their behaviour, it did not examine the perceptions of others. This is clearly an important avenue to explore as parents are more likely to respond negatively to misbehaviour if they view their child as being responsible (Johnston & Patenaude, 1994). Johnston and Leung (2001) reported that while parents attributed less control to positive behaviours while the child was on medication, medication increased attributions of control when the behaviour was negative. The authors interpreted their findings as evidence that medication treatments could be altering attributions in a way that might, potentially lead to less positive parental responses to appropriate behaviour (Johnston & Leung,



2001). Studies examining parental attributions, however, tend to look at these factors in relation to social behaviour, thus, while the attributions made by participants in the current study did not differ between medication conditions, it may be useful to extend these investigations. Examining the effects that medication may have on the attributions made, both by parents and by teachers, in relation to children with ADHD's performance on more academic-based tasks, would allow us to determine whether the pattern detected by Johnston & Leung (2001) holds true with regards to non-social behaviours.

Lastly, as previously discussed, Barkley (1997) contends that other deficits seen in ADHD, such as working memory, self-regulation etc., result from a central impairment in response inhibition. Given the current improvements medication appears to have on the inhibitory control of children with ADHD, it would be interesting to further explore the relationships between inhibitory control and other known-deficits in ADHD in relation to the effects of medication.

## 5. Conclusion

The present research used a newly developed measure – the Animal Stroop Task (Wright et al., 2003) - to examine whether methylphenidate-based medication is effective in improving inhibitory control in children with ADHD. In addition, the study looked at children's own attributions for their level of response inhibition across a variety of variables, including medication status. Although subject to methodological limitations outlined above, the study found that methylphenidate did, indeed, improve the inhibitory control of children with ADHD. This fits with previous reports of methylphenidate ameliorating deficits in response inhibition as measured by other tests of inhibitory control (Broyd et al., 2005, Everett et al., 1991), as well as reported improvements in other areas of difficulty (Pelham et al., 1986; Miller et al., 1998; Hood et al., 2005; Kempton et al., 1999). However, despite this improvement, the study did not detect any significant differences between the inhibitory control of children with ADHD (irrespective of medication status) and children in the control group. This is in contrast to a considerable amount of evidence documenting deficits in the inhibitory control of children with ADHD, and challenges the well-established theory that poor response inhibition is the cardinal feature of ADHD (Barkley, 1997). This lack of difference, however, may provide support for contentions that different tasks tap different components of inhibition and that not all levels of inhibition are necessarily impaired in children with ADHD (Kerns et al., 2001). Alternatively, it may offer support for the 'delay aversion' hypothesis in which cognitive deficits in children with ADHD are thought to be more motivational in nature (Sonuga-Barke et al., 1996). There was no difference between

the locus of control of children with and without ADHD, and locus of control was found not to be related to inhibitory control. Additionally, children with and without ADHD did not differ in the degree to which they attributed their performance to external factors and perceptions for behaviour remained comparable, regardless of whether or not children with ADHD had received medication. Thus, contrary to popular belief (e.g. Hoza et al., 1993; Parish-Plass, 1995), children with ADHD did not appear more likely to attribute their performance to external factors or to view their experiences as being outwith their personal control. Furthermore, the results are consistent with other studies that generally fail to find any deleterious effects of medication with regards to children's attributions for their behaviour (e.g. Carlson et al., 1993); Milich et al., 1989). The thesis identified a number of areas for future research, including further exploration of the links between medication, response inhibition and behaviour, in relation to the potential influence that age and gender may have on these relationships. Children with ADHD face a number of difficulties, many of which may improve with drug-treatment. While the Animal Stroop Task may not be effective in differentiating between children with a clinical diagnosis of ADHD and normally developing children, it may provide an additional tool to monitor the effects of medication-based treatments such as methylphenidate.

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## 7. Appendices

### 7.1 Appendix: Diagnostic Criteria for ADHD

#### **Diagnostic Criteria for Attention Hyperactivity Disorder in DSM-IV**

**(American Psychiatric Association, 1994)**

Either 1 or 2:

1. Six or more of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level.

#### *Inattention:*

- often fails to give close attention to details or makes careless mistakes in school work, work or other activities
- often has difficulty sustaining attention in tasks or play activities
- often does not seem to listen when spoken to directly
- often does not follow through on instructions and fails to finish school work, chores or work duties

- often has difficulty organising tasks and activities
- often avoids or dislikes tasks that require sustained mental effort
- often loses things necessary for tasks or activities
- is often easily distracted by extraneous stimuli
- is often forgetful in daily activities

2. Six or more of the following symptoms of *hyperactivity-impulsivity* have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level.

*Hyperactivity:*

- often fidgets with hands or feet or squirms in seat
- often leaves seat in classroom or in other situations in which remaining seated is expected
- often runs about or climbs excessively in situations in which it is inappropriate
- often has difficulty playing or engaging in leisure activities quietly
- is often on the go or acts as if driven by a motor
- often talks excessively



*Impulsivity:*

- often blurts out answer before questions have been completed
- often has difficulty awaiting turn
- often interrupts or intrudes on others

Some of these symptoms were present before the age of 7 years

Some impairment from the symptoms is present in *two or more settings* (e.g. home and school)

Clinically significant impairment in social, academic or occupational functioning

Not due to another disorder

Specify *combined type* if inattention and overactivity-impulsivity are present;  
*inattentive type* if overactivity is absent; *hyperactive-impulsive type* if inattentiveness is absent.

## **Diagnostic Criteria for Attention Hyperactivity Disorder in ICD-10**

### **(World Health Organisation, 1992)**

The cardinal features are impaired attention and overactivity. Both are necessary for the diagnosis and should be evident in *more than one situation* (e.g. home and school). *Impaired attention* is manifested by prematurely breaking off from tasks and leaving activities unfinished.

The children change frequently from one activity to another, seemingly losing interest in one task because they become diverted to another. These deficits in persistence and attention should be diagnosed only if they are excessive for the child's age and IQ. *Overactivity* implies excessive restlessness, especially in situations requiring relative calm. It may, depending upon the situation, involve the child running and jumping around, getting up from a seat when he or she was supposed to remain seated, excessive talkativeness and noisiness, or fidgeting and wriggling. The standard for judgement should be that the activity is excessive in the context of what is expected in the situation and by comparison with other children of the same age and IQ. This behavioural feature is most evident in structured, organised situations that require a high degree of behavioural self-control

The characteristic behaviour problems should be of early onset (before the age of 6 years) and long duration

Associated features include disinhibition in social relationships, recklessness in situations involving some danger, impulsive flouting of social rules, learning disorders, and motor clumsiness

Specify *hyperkinetic disorder with disturbance of activity and attention* when antisocial features of conduct disorder are absent; *hyperkinetic conduct disorder* when criteria for both conduct disorder and hyperkinetic disorder are met

## 7.2 Appendix: Ethical Approval Letter

**Borders Research Ethics Committee**

NHS Borders  
Newstead  
Melrose  
Roxburghshire  
TD6 9DB

Telephone: 01896 825520  
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10 February 2006

Miss Rachel A Brackenridge  
Trainee Clinical Psychologist  
NHS Borders  
Andrew Lang Unit  
Viewfield Lane  
Selkirk  
TD7 4LJ

Dear Miss Brackenridge

**Full title of study:** An Examination of the Effects of Stimulant Medication on Actual and Self-Perceived Response Inhibition in Children with Attention Deficit Hyperactivity Disorder

**REC reference number:** 05/S0301/11

The Research Ethics Committee reviewed the above application at the meeting held on 06 February 2006.

**Ethical opinion**

The members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation.

**Conditions of approval**

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

**Approved documents**

The documents reviewed and approved at the meeting were:

Document	Version	Date
Application	V2	1 <sup>st</sup> February 2005
Investigator CV	V1	21 December 2005
Protocol	V1	21 December 2005
Letter of invitation to participant	V2	1 <sup>st</sup> February 2005
Participant Information Sheet	V2	1 <sup>st</sup> February 2005
Response to Request for Further Information		1 <sup>st</sup> February 2005

**Membership of the Committee**

The members of the Ethics Committee who were present at the meeting are listed on the attached sheet.

**Statement of compliance**

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

<b>05/S0301/11</b>	<b>Please quote this number on all correspondence</b>
--------------------	---

With the Committee's best wishes for the success of this project

Yours sincerely



**Chair**

Email: [deborah.adams@borders.scot.nhs.uk](mailto:deborah.adams@borders.scot.nhs.uk)

Enclosures:                      *List of names and professions of members who were present at the meeting and those who submitted written comments*  
   *Standard approval conditions*

### 7.3 Appendix: Participant Invitation Letter

Rachel Brackenridge  
Trainee Clinical Psychologist  
CAMHS

Telephone:

Email:

Dear Parent/Guardian

We are conducting a research project looking at response inhibition in children with Attention Deficit Hyperactivity Disorder (ADHD).

I would like to invite your child to participate in the project.

I have enclosed some information to help you decide whether or not you would like your child to take part. I have also enclosed some information for your child. If they have any problems reading it, please read it aloud to him/her.

If you decide to participate, please sign the enclosed consent forms and return them in the enclosed SAE. My contact details are also enclosed; please do not hesitate to contact me with any questions or queries. You are not in any way obliged to take part in this project and can withdraw at any time without giving a reason.

I look forward to hearing from you.

Yours sincerely

Rachel Brackenridge  
Trainee Clinical Psychologist

## 7.4 Appendix: Parent Information Sheet

### 7.4.1 Experimental Group

#### **Parent/Guardian Information Sheet**

I am conducting a study as part of my qualification in clinical psychology, working with children with and without a diagnosis of Attention Deficit Hyperactivity Disorder (ADHD), to look more closely at their actual- and self- perceived level of response inhibition.

The title of the project is: *An examination of the effects of stimulant medication on actual- and self- perceived response inhibition: A comparison between children with and without attention deficit hyperactivity disorder.*

#### **What is response inhibition?**

Response inhibition refers to the human skill which allows us to prevent ourselves from doing things automatically, or avoid the usual routine action, e.g. response inhibition allows us to be polite when we are given an unwanted present.

Most children develop the ability to inhibit their responses and learn to control their actions as they get older. However, some continue to have difficulties with such skills, leading them to act without thinking, blurt out comments inappropriately and/or appear overactive and distractible. Amongst other symptoms, this kind of behaviour is often characteristic of children with ADHD.

Your child is being invited to take part in this research study. Before you decide whether or not you would like them to take part, it is important for you to understand why the research is being carried out and what it will involve. Please take the time to read the following information carefully and discuss it with others if you wish. Please contact me if there is



anything that is not clear or if you would like further information. Thank you for reading this.

**Why has my child been chosen?**

I am inviting children with a diagnosis of ADHD and children without a diagnosis. I am inviting children without a diagnosis, as a control group, so I can compare the two groups and see if there are any differences.

**What is the purpose of the study?**

The aim of the study is to look at children's actual and self-perceived levels of response inhibition. I would like to compare children who have ADHD and children who do not. I would also like to see whether medication has a direct effect on such abilities in children with ADHD. Currently, the research in this area has produced unclear results.

**What does the study involve?**

Your child will be asked to complete a brief test of intelligence (involves creating patterns with a series of blocks and then giving the meanings of numerous everyday words), a computerised test of response inhibition (children are asked to look at pictures where the head of one of four animals has been swapped with another animal. The child is asked to give the name for the "body" of the animal, thus having to inhibit the more prominent response based on the face). Your child will then be asked to complete a short questionnaire and asked a few additional questions about how they felt they got on in the computer task. This will take approximately 60 minutes in total. These will be carried out wherever is most convenient for you: in school or at the Andrew Lang Unit in Selkirk. In some cases, it may be possible to visit your child at home. If this is preferable, please feel free to contact the researcher directly or to indicate this on the consent form.

Following this session, I will arrange to meet with your child for a second time. During this session, your child will be asked to complete the same computerised task and the same series of questions. This will be a shorter session taking only 30-40 minutes.

On one of these two occasions, you will be asked not to give your child their usual ADHD medication before meeting with me. Missing this one dose of medication will not be harmful to your child and they will be able to take their medication soon after completing the assessment. If you are concerned about this possibility, please do contact the Andrew Lang Unit to discuss this with either myself or Dr ..... (Child & Adolescent Psychiatrist). Your GP will also be notified about your child's participation in the study as a routine measure.

**Will I find out how it went?**

If you wish to hear about the results of the study, you can request a written or verbal summary of the results.

**Do I have to take part?**

You do not have to take part. Even if you sign the consent form, you can withdraw at any time without giving a reason. Your child will be told at the start of testing, and reminded throughout testing, that they can leave at any time without giving a reason. A decision to withdraw, or a decision not to take part, will not affect the standard of care you or your child receive.

**Will there be any disadvantages?**

Although unlikely, should your child become unhappy during the study, the testing will be stopped immediately. Your child will be asked throughout the study if they are happy to continue and they will be reminded that they can leave at any time without giving a reason.

**What about confidentiality?**

All of the information in the study is confidential. The study is designed to promote knowledge about ADHD, but may be of no direct benefit to you and your child.

If you have no objections to your child's participation in this project, please sign the enclosed consent form and return it in the SAE provided.

**What should I do if I want my child to take part?**

If you decide that you would like your child to take part, then please sign the consent form and send it back in the envelope provided. I will then be in touch via mail or telephone to arrange the details of where you would like me to see your child, either at school or at the Andrew Lang Unit in Selkirk. In some cases, it may be possible to visit your child at home. If this is preferable, please feel free to contact the researcher directly or to indicate this on the consent form. I will arrange the session times in collaboration with yourselves and also answer any questions you might have.

Thank you for taking the time to consider taking part in this study. If you have any questions or wish to discuss any aspects of the study, please do not hesitate to contact me. My contact details are given at the top of this letter.

Yours sincerely

Rachel Brackenridge

Trainee Clinical Psychologist

## 7.4.2 Control Group

### **Parent/Guardian Information Sheet**

I am conducting a study as part of my qualification in clinical psychology, working with children with and without a diagnosis of Attention Deficit Hyperactivity Disorder (ADHD), to look more closely at their actual- and self-perceived level of response inhibition.

The title of the project is: *An examination of the effects of stimulant medication on actual- and self- perceived response inhibition: A comparison between children with and without attention deficit hyperactivity disorder.*

#### **What is response inhibition?**

Response inhibition refers to the human skill which allows us to prevent ourselves from doing things automatically, or avoid the usual routine action, e.g. response inhibition allows us to be polite when we're given an unwanted present.

Most children develop the ability to inhibit their responses and learn to control their actions as they get older. However, some continue to have difficulties with such skills, leading them to act without thinking, blurt out comments inappropriately and/or appear overactive and distractible. Amongst other symptoms, this kind of behaviour is often characteristic of children with ADHD.

Your child is being invited to take part in this research study. Before you decide whether or not you would like them to take part, it is important for you to understand why the research is being carried out and what it will involve. Please take the time to read the following information carefully and discuss it with others if you wish. Please contact me if there is anything that is not clear or if you would like further information. Thank you for reading this.

**Why has my child been chosen?**

I am inviting children with a diagnosis of ADHD and children without a diagnosis. I am inviting children without a diagnosis, as a control group, so I can compare the two groups and see if there are any differences.

**What is the purpose of the study?**

The aim of the study is to look at children's actual and self-perceived levels of response inhibition. I would like to compare children who have ADHD and children who do not. I would also like to see whether medication has a direct effect on such abilities in children with ADHD. Currently, the research in this area has produced unclear results.

**What does the study involve?**

Your child will be asked to complete a brief test of intelligence (involves creating patterns with a series of blocks and then giving the meanings of numerous everyday words), a computerised test of response inhibition (children are asked to look at pictures where the head of one of four animals has been swapped with another animal. The child is asked to give the name for the "body" of the animal, thus having to inhibit the more prominent response based on the face). Your child will then be asked to complete a short questionnaire and asked a few additional questions about how they felt they got on in the computer task. This will take approximately 60 minutes in total. These will be carried out wherever is most convenient for you: in school or at the Andrew Lang Unit in Selkirk. In some cases, it may be possible to visit your child at home. If this is preferable, please feel free to contact the researcher directly or to indicate this on the consent form.

**Will I find out how it went?**

If you wish to hear about the results of the study, you can request a written or verbal summary of the results.

**Do I have to take part?**

You do not have to take part. Even if you sign the consent form, you can withdraw at any time without giving a reason. Your child will be told at the start of testing, and reminded throughout testing, that they can leave at any time without giving a reason. A decision to withdraw, or a decision not to take part, will not affect the standard of care you or your child receive.

**Will there be any disadvantages?**

Although unlikely, should your child become unhappy during the study, the testing will be stopped immediately. Your child will be asked throughout the study if they are happy to continue and they will be reminded that they can leave at any time without giving a reason.

**What about confidentiality?**

All of the information in the study is confidential. The study is designed to promote knowledge about ADHD, but may be of no direct benefit to you and your child.

If you have no objections to your child's participation in this project, please sign the enclosed consent form and return it in the SAE provided.

**What should I do if I want my child to take part?**

If you decide that you would like your child to take part, then please sign the consent form and send it back in the envelope provided. I will then be in touch via mail or telephone to arrange the details of where you would like me to see your child, either at school or at the Andrew Lang Unit in Selkirk. In some cases, it may be possible to visit your child at home. If this is preferable, please feel free to contact the researcher directly or to indicate this on the consent form. I will arrange the session time in collaboration with yourselves and also answer any questions you might have.

Thank you for taking the time to consider taking part in this study. If you have any questions or wish to discuss any aspects of the study, please do not hesitate to contact me. My contact details are given at the top of this letter.

Yours sincerely

Rachel Brackenridge

Trainee Clinical Psychologist

## 7.5 Appendix: Child Information Sheet

### 7.5.1 Experimental Group

#### Information Sheet for Children

Would you like to take part in a project we are doing at the moment? Please read this sheet or ask someone to read it to you, to help you make up your mind.

I would like to meet with you to do some puzzles, some computer games and to ask you some questions. This will take about 1 hour. After that I would like you to come back another time to try the computer game again. This time we will only meet for about 30-40 minutes. One of these times, you will be asked not to take your medication before meeting with me. The person who looks after you will tell you when not to take your medicine.

All the information gathered will be private.

You don't have to take part if you don't want to.

If you would like to take part, but then change your mind later, that's okay. No one will mind.



Your parent(s)/guardian(s) have been told about the project and you can talk to them about it. If you would like to talk to me before you make up your mind, we can sort that out.

Rachel Brackenridge

Trainee Clinical Psychologist

### 7.5.2 Control Group

#### Information Sheet for Children

Would you like to take part in a project we are doing at the moment? Please read this sheet or ask someone to read it to you, to help you make up your mind.

I would like to meet with you to do some puzzles, some computer games and to ask you some questions. This will take about 1 hour.

All the information gathered will be private.

You don't have to take part if you don't want to.

If you would like to take part, but then change your mind later, that's okay. No one will mind.

Your parent(s)/guardian(s) have been told about the project and you can talk to them about it. If you would like to talk to me before you make up your mind, we can sort that out.

Rachel Brackenridge

Trainee Clinical Psychologist

## 7.6 Appendix: Parent Consent Form

### CONSENT FORM

#### CONSENT BY PARENT/GUARDIAN FOR THEIR CHILD TO PARTICIPATE IN:

Title: -

An examination of the effects of stimulant medication on actual- and self- perceived response inhibition: A comparison between children with and without attention deficit hyperactivity disorder.

Researcher: -

Rachel Brackenridge, Trainee Clinical Psychologist.

Name of  
child:.....

Name of  
Parent/Guardian:.....

Address:.....  
...

.....  
.

Contact telephone  
number.....

I confirm that I have read and understood the information sheet for the above study and have had the opportunity to contact Rachel Brackenridge, Trainee Clinical Psychologist, to ask questions.

I have agreed to my child taking part in the study as it has been outlined to me.

I understand that these assessments are part of a research project designed to promote knowledge regarding ADHD, which has been approved by the Borders NHS Ethics Committee, and may be of no benefit to me personally.

I understand that my child's participation is voluntary and that I am free to withdraw my child at any time, without giving a reason, without my child's medical care being affected.

I hereby fully and freely consent to my child participating in the study, which is outlined on the enclosed information sheet.

Signature of  
Parent/Guardian:.....Date:.....  
...

Signature of  
Researcher:.....Date:.....  
...

I wish my child to be seen (please tick box)

At school ☐

At the Andrew Lang Unit ☐

7.7 Appendix: Child Consent Form

CONSENT FORM FOR CHILDREN

(Please circle your answer)

Have you read the project information sheet or had someone read it to you?

Yes

No

Have you been able to ask questions about the project?

Yes

No

Do you understand that you don't have to take part in the project if you don't want to?

Yes

No

Do you understand that even if you say yes now, you can change your mind at any time and you don't have to give a reason?

Yes

No

Do you want to take part in this project?

Yes

No

Your name..... Researcher's Name.....

Your signature..... Researcher's Signature.....

Date..... Date.....

## 7.8 Appendix: Animal Stroop Task Instructions

### Task Instructions

An introductory picture of a hot air balloon appears. Explain:

*If I asked you to name this picture, you'd say...* (wait for the child to respond), then say...

*That's right, balloon/hot air balloon*

*Now you'll see some more pictures and I'd like you to name each picture as quickly as you can without making too many mistakes.*

Press space bar to continue

6 warm-up pictures are shown to get kids used to responding as quickly as they can.

*You are now going to see some pictures. I would like you to tell me the names of these pictures as quickly as possible.*

*Can you name the animals on this card for me?* (show card of 4 animals).

Run through these animal names twice.

### Incongruent Blocks

(Cat's head on horse's body)

*You're about to see some strange pictures where the animal's head is different to its body. Sometimes the animal will be wearing a different animal's head like this one, and sometimes it will have a cartoon face for a head. Your job is to name the animal's body as quickly as you can.*

*Now can you name the body of this funny animal?*

If children give the correct answer, they should be allowed to proceed with the test.

Incorrect answers should be followed by more direct prompts such as,

*I want you to name the body of the funny animal*

Followed by further repetition of the instructions before proceeding with the test.

Matching Blocks

*You're about to see some pictures of animals on the screen. I'd like you to name the animal as quickly as you can after it appears.*

Incongruent Blocks

(Cat's head on horse's body)

*You're about to see some more strange pictures where the animal's head is different to its body. Sometimes the animal will be wearing a different animal's head like this one, and sometimes it will have a cartoon face for a head. Your job is to name the animal's body as quickly as you can.*

*Now can you name the body of this funny animal?*



# 7.9 Appendix: Animal Stroop Scoring Sheet

Name: \_\_\_\_\_

Filename: \_\_\_\_\_

Stroop version: WORD / ANIMAL

Date: \_\_\_\_\_

A/B					A/B					A/B				
	Res	M	VK	ART		Res	M	VK	ART		Res	M	VK	ART
1					1					25				
2					2					26				
3					3					27				
4					4					28				
5					5					29				
6					6					30				
7					7					31				
8					8					32				
9					9					33				
10					10					34				
11					11					35				
12					12					36				
13					13					37				
14					14					38				
15					15					39				
16					16					40				
17					17					41				
18					18					42				
19					19					43				
20					20					44				
21					21					45				
22					22					46				
23					23					47				
24					24					48				

Res = Response, M = Manual timing error, VK = Voice detection error, Art = Articulation error

## 7.10 Appendix: Self-Evaluation and Attribution Questionnaire

### 7.10.1 Experimental Group

#### Attribution Measure

Before Animal Stroop Test:

***Do you think you'll do a good or bad job on the Animal Stroop Task?***

Good

Bad

***How many do you think you'll get correct? (mark out of 10)***

Following completion of the Animal Stroop Task:

***Did you do a good or bad job on the task?***

Good

Bad

***How many do you think you got correct? (mark out of 10)***

Participants will then be asked to rate (using a scale from 0-10) the degree to which each of five attribution categories was responsible for his/her success/failure:

How easy was it to pay attention?



1 2 3 4 5 6 7 8 9 10

Not at all easy

Quite easy

Very Easy

How hard did you try?



1 2 3 4 5 6 7 8 9 10

Not at all hard

Quite hard

Very hard

How hard did you think the task was?



1 2 3 4 5 6 7 8 9 10

Not at all hard

Quite hard

Very hard

How lucky were you?



1 2 3 4 5 6 7 8 9 10

Not at all lucky

Quite lucky

Very lucky

Group 1 participants in the ‘medicated’ condition will be asked an additional question:

*How much do you think your medication helped you on the computer task?*

1	2	3	4	5	6	7	8	9	10
Not at all helpful				Quite helpful			Very helpful		

Lastly, participants will then be asked to rank these five factors in order from most to least important.

*Rank Importance of these factors:*

Attention	1	-	most important
Tried Hard	2		
Task Difficulty	3		
Luck	4		
Medicine	5	-	least important

*How do you think you would have got on with the task if you’d taken your medicine as usual?*

Better	The Same	Worse
--------	----------	-------

### 7.10.2 Control Group

#### Attribution Measure

Before Animal Stroop Test:

***Do you think you'll do a good or bad job on the Animal Stroop Task?***

Good

Bad

***How many do you think you'll get correct?*** (mark out of 10)

Following completion of the Animal Stroop Task:

***Did you do a good or bad job on the task?***

Good

Bad

***How many do you think you got correct?*** (mark out of 10)

Participants will then be asked to rate (using a scale from 0-10) the degree to which each of four attribution categories was responsible for his/her success/failure:

How easy was it to pay attention?

1	2	3	4	5	6	7	8	9	10
Not at all easy			Quite easy				Very Easy		

How hard did you try?

1	2	3	4	5	6	7	8	9	10
Not at all hard			Quite hard				Very hard		

How hard did you think the task was?

1	2	3	4	5	6	7	8	9	10
Not at all hard			Quite hard				Very hard		

How lucky were you?

1	2	3	4	5	6	7	8	9	10
Not at all lucky			Quite lucky				Very lucky		

Lastly, participants will then be asked to rank these four factors in order from most to least important.

***Rank Importance of these factors:***

Attention	1	-	most important
Tried Hard	2		
Task Difficulty	3		
Luck	4	-	least important

7.11 Appendix: Appointment Confirmation Letter

**Mrs B**

Date  
Your Ref  
Our Ref  
CHI No

Enquiries to  
Extension  
Direct Line  
Email

Dear Mrs B

Following our telephone discussion, I write to confirm the times that I will be meeting with AB at the ..... Unit

My first appointment with AB is:

**Friday 4<sup>th</sup> August at 9.30am**

On this occasion AB should **not** take his morning dose of medication. I would be grateful if you could give AB the attached slip to bring to me on the morning of the 4<sup>th</sup> of August so I can confirm that he has not had any medication.

I will be finished meeting with AB by approximately 10am and he will be able to take his morning dose of medication then. He should return to his usual medication routine the following day.



My second appointment to see AB at the ..... Unit is:

**Friday 11<sup>th</sup> August at 9.30am**

AB should take all medication as normal on this day.

I hope this is acceptable to you. If you have any queries please do not hesitate to contact me at the ..... Unit. I look forward to meeting AB soon.

Yours sincerely

Rachel Brackenridge

Trainee Clinical Psychologist

**TO BE RETURNED TO RACHEL BRACKENRIDGE**

**Name:** AB

**Date:** Friday 4<sup>th</sup> August 2006

**I can confirm that the above named child has not taken their routine morning dose of medication today and has therefore received no medication since yesterday.**

**Signature of Parent/Guardian.....**

## 7.12 Appendix: GP Letter – Notification of Patient Participation

Rachel Brackenridge  
Trainee Clinical Psychologist

Telephone:

Email:

Date

Dear Dr .....

My name is Rachel Brackenridge and I am a Trainee Clinical Psychologist currently on placement at the ..... Unit. In conjunction with the Borders ADHD Team and Child and Family Mental Health Service, I am currently undertaking a doctorate thesis research entitled: *An examination of the effects of stimulant medication on actual- and self- perceived response inhibition: A comparison between children with and without attention deficit hyperactivity disorder.*

I am writing to inform you that one of your patients, ..... (DOB:.....), has agreed to participate in the study. As such he/she will be required to miss a single dose of his/her stimulant medication prior to meeting with me. Due to the relatively short half-life of this medication, we do not anticipate any adverse effects from this and ..... will be able to take his/her medication shortly after completing the test session. Naturally both ..... and his/her parents/guardians have consented to this aspect of the study and both myself and Dr ..... are available for consultation throughout the duration of the study. I enclose a copy of the participant information sheet for your interest.

Please do not hesitate to contact me if you have any queries.

Yours sincerely

Rachel Brackenridge  
Trainee Clinical Psychologist

### 7.13 Appendix: Participant Thank You Letter

Date  
Your Ref  
Our Ref  
CHI No  
  
Enquiries to  
Extension  
Direct Line  
Email

Dear .....

I just wanted to write and say thank you very much for taking part in my study. You did very well in all the tasks that we did and I really appreciate your help.

I plan to have finished my study (Entitled: *An examination of the effects of stimulant medication on actual- and self- perceived response inhibition: A comparison between children with and without attention deficit hyperactivity disorder*) by February 2007 and I am more than happy to let you and your parents/guardians read a summary of the results. If you would like to do this, please do not hesitate to contact me at the ..... Unit.

I hope you enjoy the rest of your summer holidays.

Best wishes,

Rachel Brackenridge  
Trainee Clinical Psychologist



## Descriptives - Experimental Group

Descriptive Statistics - Experimental Group

	N	Minimum	Maximum	Mean
	Statistic	Statistic	Statistic	Statistic
AGE	21	6.00	14.00	9.3333
FSIQ	21	62.00	144.00	94.2857
Locus of Control	21	22.50	67.50	46.7390
RT-med.Incongruent	21	676.00	1832.00	1139.6667
RT-med.Matching	21	662.00	1536.00	918.0476
RT-med.Control	21	636.00	1826.00	1102.2857
RT.Difference-med	21	-190.00	216.00	37.3810
Error-med.total	21	.00	15.00	3.1429
omissions-med.total	21	.00	25.00	2.0476
RT-unmed.Incongruent	21	792.00	1770.00	1251.7619
RT-unmed.Matching	21	599.00	1825.00	1064.3810
RT-unmed.Control	21	701.00	1836.00	1147.9048
RT.Difference-unmed	21	-66.00	298.00	103.8571
Error-unmed.total	21	.00	14.00	3.4762
Omissions-unmed.total	21	.00	16.00	2.6667
Valid N (listwise)	21			

Descriptive Statistics - Experimental Group

	Std.	Skewness		Kurtosis	
	Statistic	Statistic	Std. Error	Statistic	Std. Error
AGE	2.57553	.290	.501	-1.153	.972
FSIQ	18.16079	.829	.501	1.678	.972
Locus of Control	11.93634	.112	.501	-.654	.972
RT-med.Incongruent	301.54027	.801	.501	.277	.972
RT-med.Matching	258.27340	1.358	.501	1.231	.972
RT-med.Control	312.77886	.839	.501	.229	.972
RT.Difference-med	85.30679	-.436	.501	1.754	.972
Error-med.total	3.38062	2.379	.501	7.119	.972
omissions-med.total	5.81787	3.560	.501	13.249	.972
RT-unmed.Incongruent	285.68460	.062	.501	-.922	.972
RT-unmed.Matching	290.12264	.785	.501	1.065	.972
RT-unmed.Control	283.19550	.594	.501	.246	.972
RT.Difference-unmed	103.01616	.391	.501	-.345	.972
Error-unmed.total	3.84212	1.900	.501	3.164	.972
Omissions-unmed.total	4.56435	1.839	.501	2.737	.972
Valid N (listwise)					

## Appendix 7.14

## Descriptive Statistics - Attribution Questionnaire Responses - Experimental Group

	N	Minimum	Maximum	Mean
	Statistic	Statistic	Statistic	Statistic
Rate.Before-med	21	5.00	10.00	8.8571
Rate.After-med	21	5.00	10.00	8.8571
Attention-med	21	2.00	10.00	8.1905
Trying-med	21	6.00	10.00	8.9524
Task.Difficulty-med	21	1.00	9.00	3.0476
Luck-med	21	1.00	10.00	8.0476
Attention.Rank-med	21	1.00	5.00	2.3333
Try.Rank-med	21	1.00	5.00	2.4762
Difficulty.Rank-med	21	1.00	5.00	3.7619
Luck.Rank-med	21	1.00	5.00	3.5714
Medication.Rank-med	21	1.00	5.00	2.7143
Medication.Help	21	1.00	10.00	7.0952
If.Medication.Taken	21	.00	2.00	1.2857
RateBefore-unmed	21	.00	10.00	7.1905
RateAfter-unmed	21	6.00	10.00	9.1429
Attention-unmed	21	2.00	10.00	6.9524
Trying-unmed	21	3.00	10.00	8.4286
TaskDifficulty-unmed	21	1.00	10.00	3.5238
Luck-unmed	21	1.00	10.00	6.7143
Attention.Rank-unmed	21	1.00	4.00	2.0476
Try.Rank-unmed	21	1.00	5.00	2.0952
Difficulty.Rank-unmed	21	1.00	5.00	3.6667
Luck.Rank-unmed	21	2.00	5.00	3.8571
Medication.Rank-unmed	21	1.00	5.00	3.3333
Valid N (listwise)	21			

**Descriptive Statistics - Attribution Questionnaire Responses - Experimental Group**

	Std.	Skewness		Kurtosis	
	Statistic	Statistic	Std. Error	Statistic	Std. Error
Rate.Before-med	1.49284	-1.227	.501	.861	.972
Rate.After-med	1.79682	-1.536	.501	1.007	.972
Attention-med	2.46209	-1.509	.501	1.318	.972
Trying-med	1.56449	-1.126	.501	-.425	.972
Task.Difficulty-med	2.78345	1.330	.501	.468	.972
Luck-med	2.57830	-1.451	.501	1.665	.972
Attention.Rank-med	1.35401	.792	.501	-.557	.972
Try.Rank-med	1.12335	.533	.501	-.263	.972
Difficulty.Rank-med	1.44585	-.747	.501	-.884	.972
Luck.Rank-med	1.28730	-.641	.501	-.425	.972
Medication.Rank-med	1.38358	.069	.501	-1.281	.972
Medication.Help	3.33024	-.801	.501	-.929	.972
If.Medication.Taken	.64365	-.330	.501	-.510	.972
RateBefore-unmed	2.99364	-.734	.501	-.245	.972
RateAfter-unmed	1.31475	-1.456	.501	1.215	.972
Attention-unmed	2.99126	-.110	.501	-1.819	.972
Trying-unmed	2.27093	-1.129	.501	-.017	.972
TaskDifficulty-unmed	3.09223	1.124	.501	-.033	.972
Luck-unmed	3.24257	-.553	.501	-.938	.972
Attention.Rank-unmed	.92066	.324	.501	-.886	.972
Try.Rank-unmed	1.13599	.928	.501	.515	.972
Difficulty.Rank-unmed	1.46059	-.631	.501	-1.121	.972
Luck.Rank-unmed	.79282	-.394	.501	.154	.972
Medication.Rank-unmed	1.55991	-.356	.501	-1.423	.972
Valid N (listwise)					

**Descriptives - Control Group****Descriptive Statistics - Control Group**

	N	Minimum	Maximum	Mean
	Statistic	Statistic	Statistic	Statistic
AGE	25	6.00	13.00	9.2000
FSIQ	25	71.00	132.00	102.4800
Locus.of.Control	25	25.00	65.38	44.0844
RT.Incongruent	25	714.00	1619.00	1091.9200
RT.Matching	25	557.00	1310.00	862.9600
RT.Control	25	676.00	1509.00	1018.5600
RT.Difference	25	-197.00	270.00	73.3600
Error.Total	25	.00	6.00	1.5200
Omission.Total	25	.00	4.00	.3600
Valid N (listwise)	25			



## Descriptive Statistics - Control Group

	Std.	Skewness		Kurtosis	
	Statistic	Statistic	Std. Error	Statistic	Std. Error
AGE	2.00000	.245	.464	-.955	.902
FSIQ	17.02479	-.011	.464	-.702	.902
Locus.of.Control	11.44228	.281	.464	-1.044	.902
RT.Incongruent	258.74745	.405	.464	-.800	.902
RT.Matching	188.09848	.566	.464	-.300	.902
RT.Control	234.77029	.511	.464	-.762	.902
RT.Difference	107.71602	-.248	.464	.597	.902
Error.Total	1.58430	1.293	.464	1.337	.902
Omission.Total	1.11355	3.139	.464	8.826	.902
Valid N (listwise)					

## Descriptive Statistics - Attribution Questionnaire Responses - Control Group

	N	Minimum	Maximum	Mean
	Statistic	Statistic	Statistic	Statistic
RateBefore	25	6.00	10.00	7.9200
RateAfter	25	5.00	10.00	8.9600
Attention	25	4.00	10.00	7.8000
Trying	25	4.00	10.00	8.3600
Task.Difficulty	25	1.00	9.00	3.2400
Luck	25	1.00	10.00	7.3600
Attention.Rank	25	1.00	3.00	1.5200
Try.Rank	25	1.00	3.00	1.7600
Difficulty.Rank	25	1.00	4.00	3.2400
Luck.Rank	25	2.00	4.00	3.4800
Valid N (listwise)	25			

## Descriptive Statistics - Attribution Questionnaire Responses - Control Group

	Std.	Skewness		Kurtosis	
	Statistic	Statistic	Std. Error	Statistic	Std. Error
RateBefore	1.22202	.164	.464	-.805	.902
RateAfter	1.13578	-1.956	.464	5.352	.902
Attention	2.16025	-.636	.464	-1.063	.902
Trying	1.77670	-.840	.464	-.120	.902
Task.Difficulty	2.25979	.830	.464	.006	.902
Luck	2.91376	-.850	.464	-.566	.902
Attention.Rank	.71414	1.043	.464	-.151	.902
Try.Rank	.66332	.302	.464	-.612	.902
Difficulty.Rank	.77889	-1.038	.464	1.425	.902
Luck.Rank	.71414	-1.043	.464	-.151	.902
Valid N (listwise)					

## 7.15 Appendix: ANOVA used to examine the effect of 'Order' Variable

## Univariate Analysis of Variance

### Warnings

Post hoc tests are not performed for GROUP because there are fewer than three groups.  
 Post hoc tests are not performed for ORD.CODE because there are fewer than three groups.

### Between-Subjects Factors

		Value Label	N
GROUP	1.00	adhd-on	19
	2.00	adhd-off	21
ORD.CODE	.00	off-on	21
	1.00	on-off	19

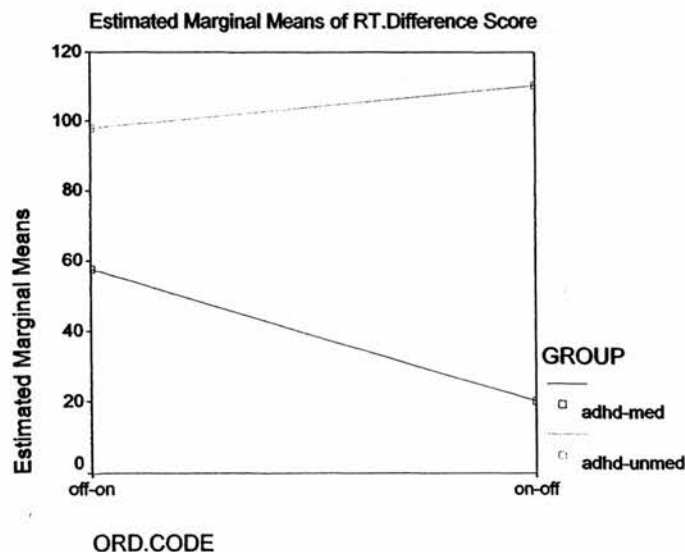
### Tests of Between-Subjects Effects

Dependent Variable: RT1.DIFF

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	48138.291 <sup>a</sup>	3	16046.097	2.166	.109
Intercept	203807.483	1	203807.483	27.514	.000
GROUP	42281.055	1	42281.055	5.708	.022
ORD.CODE	1526.884	1	1526.884	.206	.653
GROUP * ORD.CODE	6158.438	1	6158.438	.831	.368
Error	266663.709	36	7407.325		
Total	530892.000	40			
Corrected Total	314802.000	39			

a. R Squared = .153 (Adjusted R Squared = .082)

### Profile Plots



## 7.16 Appendix: Non-significant Results – Hypothesis 6 Rating Data

## Attribution Questionnaire Responses - Rating Data

### Attribution Questionnaire Responses - Experimental Group (medicated vs unmedicated conditions)

#### T-Test

##### Paired Samples Statistics - ADHD medicated vs ADHD unmedicated - Based on Transformed Data

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	RateAfter-med	2.1559	21	.24605	.05369
	RateAfter-unmed	2.2014	21	.16252	.03546
Pair 2	Attention-med	2.0317	21	.44276	.09662
	Attention-unmed	1.8327	21	.49778	.10862
Pair 3	Try-med	2.1750	21	.19587	.04274
	Try-unmed	2.0847	21	.34095	.07440
Pair 4	Task.Difficulty-med	.7615	21	.83739	.18273
	Task.Difficulty-unmed	.9013	21	.86540	.18885
Pair 5	Luck-med	1.9894	21	.54918	.11984
	Luck-unmed	1.7011	21	.77939	.17008

##### Paired Samples Correlations - ADHD medicated vs ADHD unmedicated - Based on Transformed Data

		N	Correlation	Sig.
Pair 1	RateAfter-med & RateAfter-unmed	21	.239	.297
Pair 2	Attention-med&Attention-unmed	21	.160	.489
Pair 3	Try-med&Try-unmed	21	.440	.046
Pair 4	TaskDifficulty-med&TaskDifficulty-unmed	21	.757	.000
Pair 5	Luck-med&Luck-unmed	21	.300	.186

##### Paired Samples Test - ADHD medicated vs ADHD unmedicated - Based on Transformed Data

		Paired Differences		
		Mean	Std. Deviation	Std. Error Mean
Pair 1	RateAfter-med - RateAfter-unmed	-.0455	.26046	.05684
Pair 2	Attention-med - Attention-unmed	.1990	.61107	.13335
Pair 3	Try-med - Try-unmed	.0903	.30951	.06754
Pair 4	TaskDifficulty-med - TaskDifficulty-unmed	-.1397	.59399	.12962
Pair 5	Luck-med - Luck-unmed	.2884	.80742	.17619

**Paired Samples Test - ADHD medicated vs ADHD unmedicated - Based on Transformed Data**

		Paired Differences	
		95% Confidence Interval of the Difference	
		Lower	Upper
Pair 1	RateAfter-med - RateAfter-unmed	-.1641	.0730
Pair 2	Attention-med - Attention-unmed	-.0791	.4772
Pair 3	Try-med - Try-unmed	-.0506	.2312
Pair 4	TaskDifficulty-med - TaskDifficulty-unmed	-.4101	.1306
Pair 5	Luck-med - Luck-unmed	-.0792	.6559

**Paired Samples Test - ADHD medicated vs ADHD unmedicated - Based on Transformed Data**

		t	df	Sig. (2-tailed)
Pair 1	RateAfter-med - RateAfter-unmed	-.801	20	.433
Pair 2	Attention-med - Attention-unmed	1.492	20	.151
Pair 3	Try-med - Try-unmed	1.337	20	.196
Pair 4	TaskDifficulty-med - TaskDifficulty-unmed	-1.078	20	.294
Pair 5	Luck-med - Luck-unmed	1.637	20	.117

### Attribution Questionnaire Responses - Experimental Group (medicated condition) and Controls

#### T-Test

**Group Statistics -ADHD medicated vs. Controls - Based on Transformed Data**

GROUP		N	Mean	Std. Deviation	Std. Error Mean
RateAfter	adhd	21	2.1559	.24605	.05369
	control	25	2.1832	.14919	.02984
Attention	adhd	21	2.0317	.44276	.09662
	control	25	2.0095	.32009	.06402
Trying	adhd	21	2.1750	.19587	.04274
	control	25	2.0975	.24464	.04893
TaskDifficulty	adhd	21	.7615	.83739	.18273
	control	25	.9202	.75312	.15062
Luck	adhd	21	1.9894	.54918	.11984
	control	25	1.8693	.59848	.11970

**T-Test****Independent Samples Test - ADHD medicated vs Controls - Based on Transformed Data**

		Levene's Test for Equality of Variances	
		F	Sig.
RateAfter	Equal variances assumed Equal variances not assumed	5.256	.027
Attention	Equal variances assumed Equal variances not assumed	.284	.597
Trying	Equal variances assumed Equal variances not assumed	.831	.367
TaskDifficulty	Equal variances assumed Equal variances not assumed	.454	.504
Luck	Equal variances assumed Equal variances not assumed	.745	.393

## Independent Samples Test - ADHD medicated vs Controls - Based on Transformed Data

		t-test for Equality of Means			
		t	df	Sig. (2-tailed)	Mean Difference
RateAfter	Equal variances assumed	-.464	44	.645	-.0274
	Equal variances not assumed	-.445	31.738	.659	-.0274
Attention	Equal variances assumed	.197	44	.845	.0222
	Equal variances not assumed	.191	35.684	.849	.0222
Trying	Equal variances assumed	1.170	44	.248	.0775
	Equal variances not assumed	1.193	43.916	.239	.0775
TaskDifficulty	Equal variances assumed	-.676	44	.502	-.1587
	Equal variances not assumed	-.670	40.738	.507	-.1587
Luck	Equal variances assumed	.704	44	.485	.1201
	Equal variances not assumed	.709	43.627	.482	.1201



**Independent Samples Test - ADHD medicated vs Controls - Based on Transformed Data**

		t-test for Equality of Means		
		Std. Error Difference	95% Confidence Interval of the Difference	
			Lower	Upper
RateAfter	Equal variances assumed	.05895	-.14616	.09145
	Equal variances not assumed	.06143	-.15252	.09781
Attention	Equal variances assumed	.11271	-.20498	.24933
	Equal variances not assumed	.11590	-.21296	.25731
Trying	Equal variances assumed	.06624	-.05599	.21102
	Equal variances not assumed	.06497	-.05343	.20846
TaskDifficulty	Equal variances assumed	.23460	-.63147	.31412
	Equal variances not assumed	.23681	-.63702	.31966
Luck	Equal variances assumed	.17068	-.22383	.46412
	Equal variances not assumed	.16938	-.22130	.46159

### **Attribution Questionnaire Responses - Experimental Group (Unmedicated Condition) and Controls T-Test**

**Group Statistics - ADHD unmedicated vs controls - Based on Transformed Data**

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
RateAfter	adhd	21	2.2014	.16252	.03546
	control	25	2.1832	.14919	.02984
Attention	adhd	21	1.8327	.49778	.10862
	control	25	2.0095	.32009	.06402
Trying	adhd	21	2.0847	.34095	.07440
	control	25	2.0975	.24464	.04893
TaskDifficulty	adhd	21	.9013	.86540	.18885
	control	25	.9202	.75312	.15062
Luck	adhd	21	1.7011	.77939	.17008
	control	25	1.8693	.59848	.11970

**Independent Samples Test - ADHD unmedicated vs Controls - Based on Transformed Data**

		Levene's Test for Equality of Variances	
		F	Sig.
RateAfter	Equal variances assumed Equal variances not assumed	1.190	.281
Attention	Equal variances assumed Equal variances not assumed	8.923	.005
Trying	Equal variances assumed Equal variances not assumed	2.150	.150
TaskDifficulty	Equal variances assumed Equal variances not assumed	.603	.442
Luck	Equal variances assumed Equal variances not assumed	.811	.373

## Independent Samples Test - ADHD unmedicated vs Controls - Based on Transformed Data

		t-test for Equality of Means			
		t	df	Sig. (2-tailed)	Mean Difference
RateAfter	Equal variances assumed	.395	44	.695	.0182
	Equal variances not assumed	.392	41.153	.697	.0182
Attention	Equal variances assumed	-1.455	44	.153	-.1768
	Equal variances not assumed	-1.403	32.990	.170	-.1768
Trying	Equal variances assumed	-.148	44	.883	-.0128
	Equal variances not assumed	-.144	35.506	.886	-.0128
TaskDifficulty	Equal variances assumed	-.079	44	.937	-.0189
	Equal variances not assumed	-.078	40.038	.938	-.0189
Luck	Equal variances assumed	-.828	44	.412	-.1682
	Equal variances not assumed	-.809	37.128	.424	-.1682

## Independent Samples Test - ADHD unmedicated vs Controls - Based on Transformed Data

		t-test for Equality of Means		
		Std. Error Difference	95% Confidence Interval of the Difference	
			Lower	Upper
RateAfter	Equal variances assumed	.04600	-.07454	.11085
	Equal variances not assumed	.04635	-.07543	.11174
Attention	Equal variances assumed	.12151	-.42173	.06805
	Equal variances not assumed	.12608	-.43336	.07969
Trying	Equal variances assumed	.08655	-.18723	.16161
	Equal variances not assumed	.08905	-.19350	.16788
TaskDifficulty	Equal variances assumed	.23861	-.49983	.46195
	Equal variances not assumed	.24156	-.50713	.46925
Luck	Equal variances assumed	.20325	-.57786	.24139
	Equal variances not assumed	.20797	-.58958	.25311

## 7.17 Appendix: Non-significant Results – Hypothesis 6 Ranking Data

## Attribution Questionnaire Ranking

### Attribution Questionnaire Ranking - Experimental Group Differences T-Test

#### Paired Samples Statistics - ADHD medicated vs ADHD unmedicated

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Attention-med	2.3333	21	1.35401	.29547
	Attention-unmed	2.0476	21	.92066	.20090
Pair 2	Trying-med	2.4762	21	1.12335	.24513
	Trying-unmed	2.0952	21	1.13599	.24789
Pair 3	Difficulty-med	3.7619	21	1.44585	.31551
	Difficulty-unmed	3.6667	21	1.46059	.31873
Pair 4	Luck-med	3.5714	21	1.28730	.28091
	Luck-unmed	3.8571	21	.79282	.17301
Pair 5	Medication-med	2.7143	21	1.38358	.30192
	Medication-unmed	3.3333	21	1.55991	.34040

#### Paired Samples Correlations - ADHD medicated vs ADHD unmedicated

		N	Correlation	Sig.
Pair 1	Attention-med & Attention-unmed	21	.508	.019
Pair 2	Trying-med & Trying-unmed	21	.119	.606
Pair 3	Difficulty-med&Difficulty-unmed	21	.268	.240
Pair 4	Luck-med&Luck-unmed	21	.133	.566
Pair 5	Medication-med&Medication-unmed	21	.185	.421

#### Paired Samples Test - ADHD medicated vs ADHD unmedicated

		Paired Differences		
		Mean	Std. Deviation	Std. Error Mean
Pair 1	Attention-med - Attention-unmed	.2857	1.18924	.25951
Pair 2	Trying-med - Trying-unmed	.3810	1.49921	.32715
Pair 3	Difficulty-med - Difficulty-unmed	.0952	1.75798	.38362
Pair 4	Luck-med - Luck-unmed	-.2857	1.41926	.30971
Pair 5	Medication-med - Medication-unmed	-.6190	1.88351	.41102

**Paired Samples Test - ADHD medicated vs ADHD unmedicated**

		Paired Differences	
		95% Confidence Interval of the Difference	
		Lower	Upper
Pair 1	Attention-med - Attention-unmed	-.2556	.8270
Pair 2	Trying-med - Trying-unmed	-.3015	1.0634
Pair 3	Difficulty-med - Difficulty-unmed	-.7050	.8955
Pair 4	Luck-med - Luck-unmed	-.9318	.3603
Pair 5	Medication-med - Medication-unmed	-1.4764	.2383

**Paired Samples Test - ADHD medicated vs ADHD unmedicated**

		t	df	Sig. (2-tailed)
Pair 1	Attention-med - Attention-unmed	1.101	20	.284
Pair 2	Trying-med - Trying-unmed	1.164	20	.258
Pair 3	Difficulty-med - Difficulty-unmed	.248	20	.806
Pair 4	Luck-med - Luck-unmed	-.923	20	.367
Pair 5	Medication-med - Medication-unmed	-1.506	20	.148

## 7.18 Appendix: Means for the Original Pre- and Post- Performance Rating Data



### Mean Pre- and Post- Performance Ratings for each Participant Group

#### Experimental Group (medicated) - Pre- and Post- Performance Ratings

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-Rating	21	5.00	10.00	8.8571	1.49284
Post-Rating	21	5.00	10.00	8.8571	1.79682
Valid N (listwise)	21				

#### Experimental Group (unmedicated) - Pre- and Post- Performance Ratings

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-Rating	21	.00	10.00	7.1905	2.99364
Post-Rating	21	6.00	10.00	9.1429	1.31475
Valid N (listwise)	21				

#### Control Group - Pre- and Post- Performance Ratings

	N	Minimum	Maximum	Mean	Std. Deviation
Pre-Rating	25	6.00	10.00	7.9200	1.22202
Post-Rating	25	5.00	10.00	8.9600	1.13578
Valid N (listwise)	25				

7.19 Appendix: 'Thermometer' Scale used to aid Participant Rating Procedure

